VOLUME 44 NUMBER 6 DECEMBER, 1957

AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

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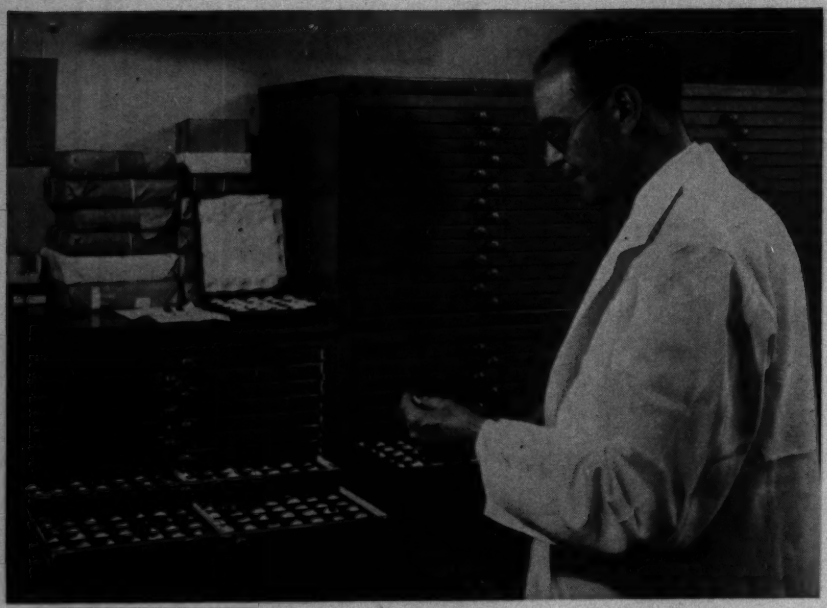
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Publication office: 450 Ahnaip St., Menasha, Wisconsin

Copyright, 1957, Ophthalmic Publishing Company, 664 North Michigan Avenue, Chicago 11, Illinois

Subscription price in United States twelve dollars yearly. In Canada and foreign countries fourteen dollars. Published monthly by the Ophthalmic Publishing Company. Subscription and Advertising Office: 664 North Michigan Avenue, Chicago 11, Illinois. Entered as second class matter at the post office at Menasha, Wisconsin. Printed in U.S.A.



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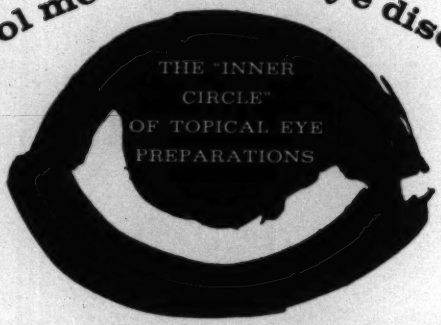
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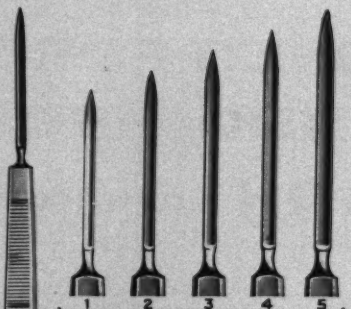
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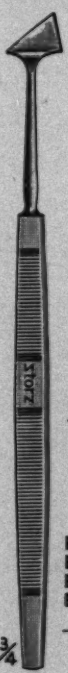
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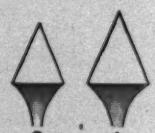
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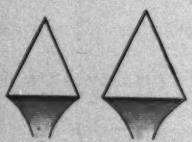
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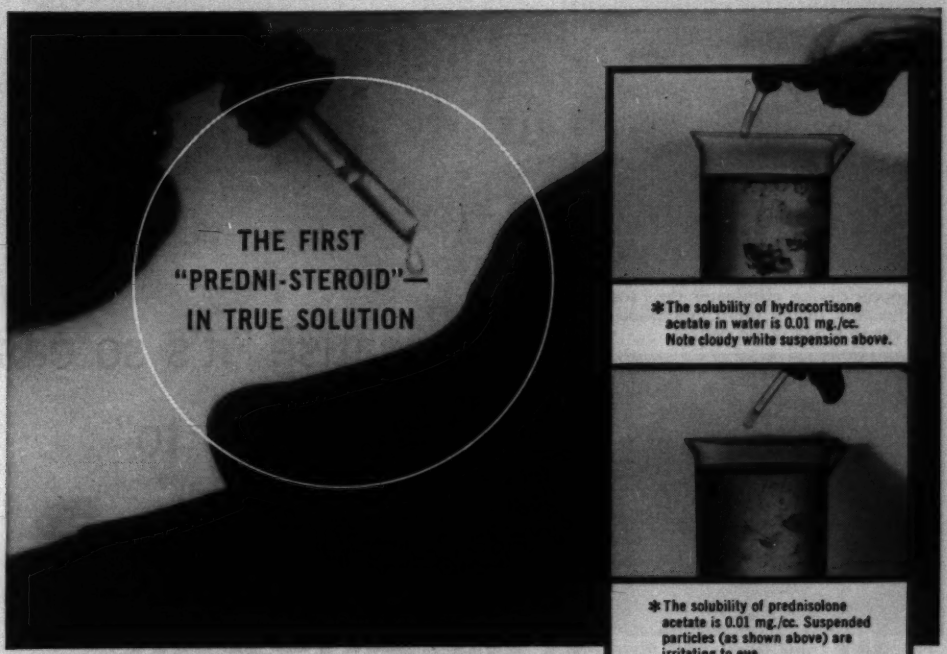


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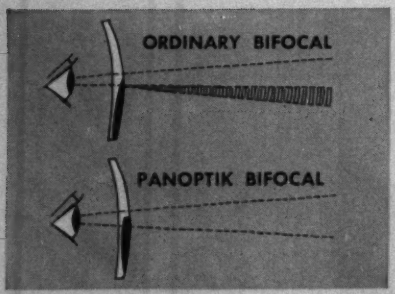
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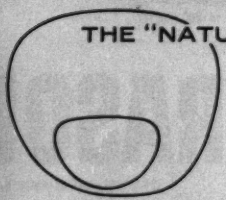


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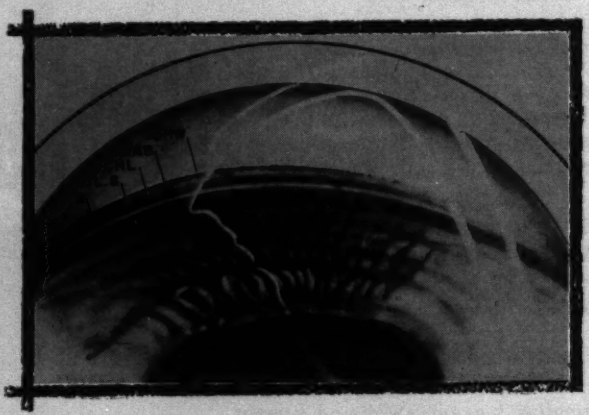
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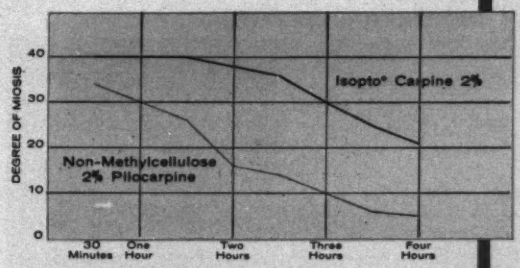
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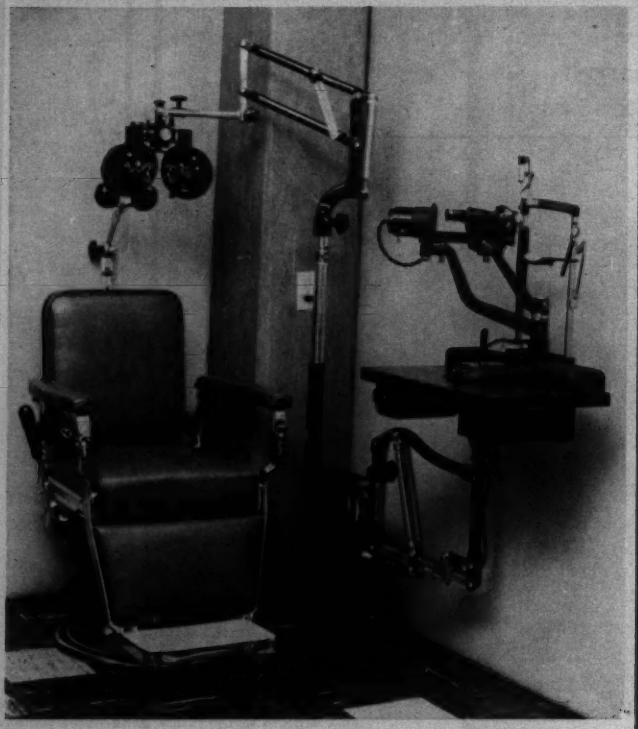
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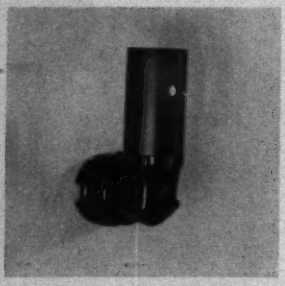


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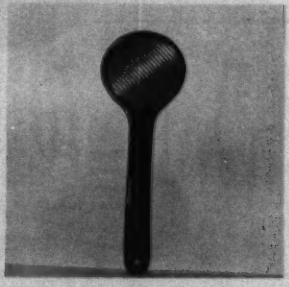
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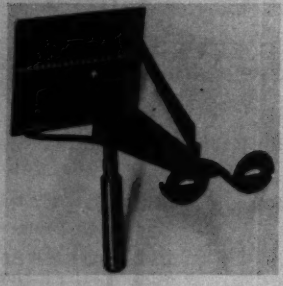
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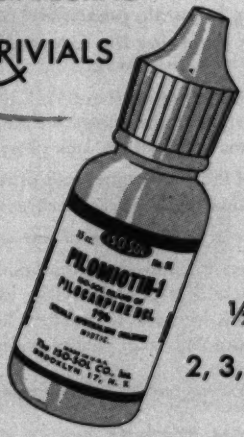
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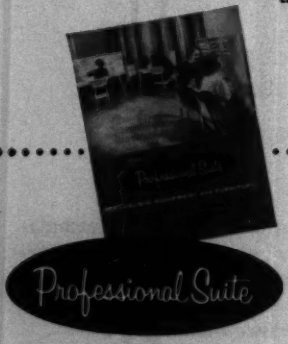
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1. *Am. J. Ophth.* 42:771, 1956.
2. *Am. J. Digest. Dis.* 22:5, 1955.
3. *Med. Times* 84:741, 1956.

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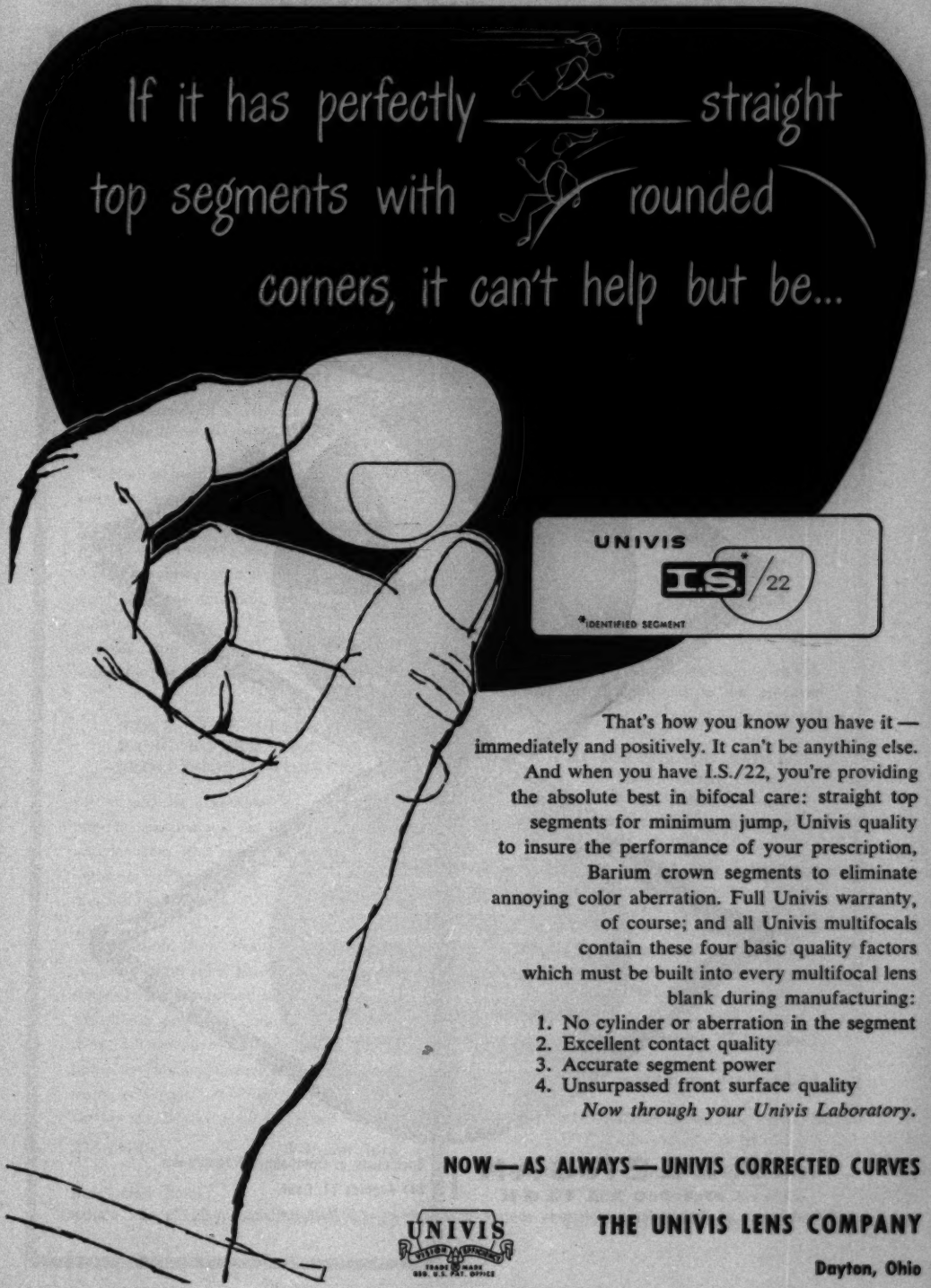
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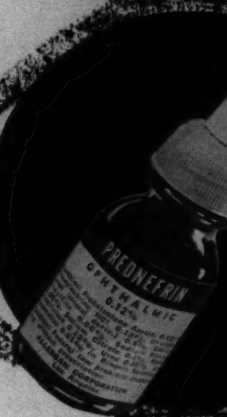
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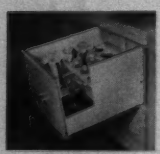


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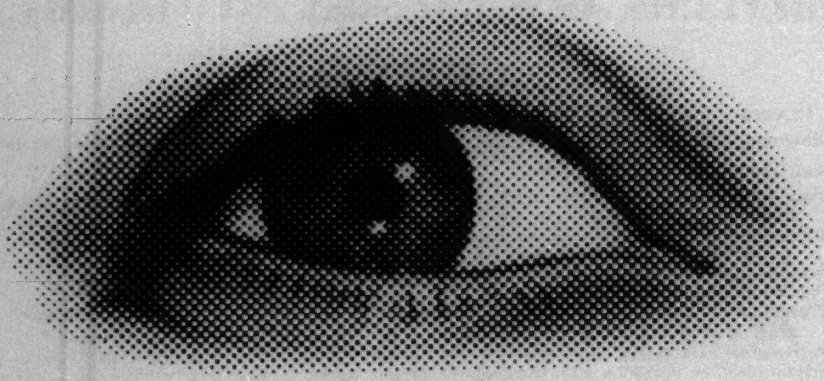
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BLOW-OUT FRACTURE OF THE ORBIT*

MECHANISM AND CORRECTION OF INTERNAL ORBITAL FRACTURE

BYRON SMITH, M.D., AND WILLIAM F. REGAN, JR., M.D.
New York

The term "blow-out" fracture refers to fracture of the orbital floor caused by a sudden increase in intraorbital pressure. This type of fracture occurs when a smooth convex object with sufficient force behind it hits the front of the orbit and forces the soft orbital contents backward without rupturing the eyeball. Blows from fists and many varieties of spheres, of the diameter of baseballs, are the most frequent causes of this type of fracture.

Several theories have been presented as to why fracture of the orbital floor can occur without concomitant fracture of the orbital rim. Pfeiffer holds that the force of the orbital contents against the floor causes fracture. LeForte, Lagrange, and others believe that the force is transmitted by bone conduction through the rigid orbital rim directly to the thin orbital floor. The fracture line is consistent with the LeForte II "line of weakness."

Pfeiffer's view, in our opinion, best explains the mechanism of internal fracture without fracture of the rim and we, therefore, employ the term "blow-out" or hydraulic fracture. The force of a convex object at the orbital entrance displaces the globe and other orbital tissues posteriorly with resultant fracture of the area of least resistance, the orbital floor. Downward displacement of the orbital contents and enophthalmos follow.

The appearance of a patient at Manhattan Eye, Ear, and Throat Hospital with "blow-out" fracture of the orbital floor stimulated our interest in the mechanism of production of this type of fracture. Our skepticism regarding direct bone transmission as a primary vector of the fracturing force is based on experimental fracture of the orbital floor in the cadaver. The case history and method of experiment follow:

CASE HISTORY

M. N., a man aged 29 years, was admitted to the Manhattan Eye, Ear, and Throat Hospital on July 29, 1956, two hours after having been struck in the right eye by a hurling ball.

Severe swelling of the right upper lid, right lower lid, and marked subconjunctival edema and hemorrhage were conspicuous. The right pupil reacted sluggishly to light. There was no abrasion of the cornea nor tear of the iris sphincter. Elevation of the right eye was limited in all fields of upward gaze and was also limited with forced duction. Marked diplopia was present in all right fields and in the primary position. A small hyphema at the base of the anterior chamber was observed.

Funduscopic examination was difficult because of the hazy media. Commotio retinae was noted in the injured eye with a reduction of the visual acuity to 20/10. Vision of the uninjured eye was 20/20. Tonometric tension was 40 mm. Hg, O.D., and 20 mm. Hg, O.S.

A small horizontal laceration of the skin extended along the lower orbital margin near its midpoint. The bony contour of the orbital margin and surface of the zygoma were intact. Exophthalmometry disclosed no evidence of ocular displacement.

X-ray films of the right orbit, skull, and nose showed no demonstrable fracture of the nasal bones. An increase of soft tissue density in the right orbit and a small amount of emphysema in the upper aspect of the right orbit were apparent. There was a depressed fracture of the floor of the right orbit; one fragment was depressed about 10 mm. (fig. 1). Measurements of the optic foramen were normal. The cranial vault and sella

* From Manhattan Eye, Ear, and Throat Hospital and the Society for Rehabilitation of the Facially Disfigured.

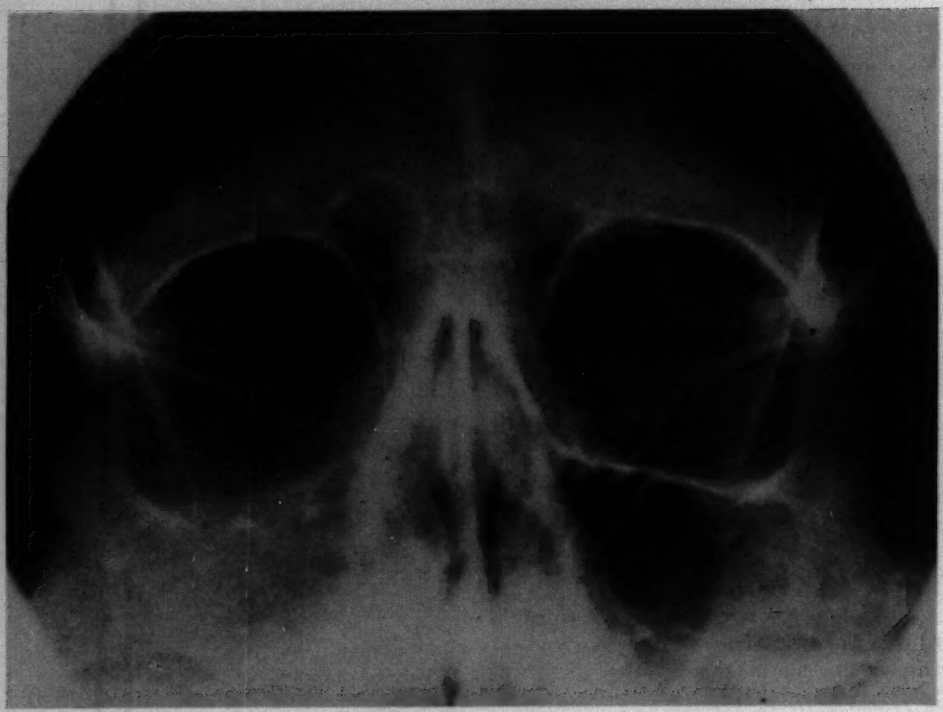


Fig. 1 (Smith and Regan). Preoperative Waters view shows depressed fracture of the orbital floor with comminuted displaced fragments of bone in the clouded right antrum.

turcica were normal. X-ray films demonstrated moderate clouding of the right ethmoids and right antrum.

On August 3, 1956, muscle measurement showed that exotropia secondary to limitation in right ocular elevation was responsible for constant diplopia. Diplopia was present in all fields of gaze (fig. 2).

By August 8th, secondary infection of the laceration and edema had sufficiently subsided to permit surgical exploration and treatment. The following procedure was performed under general anesthesia.

A skin incision 2.5 mm. in length parallel to the right lower orbital margin was extended through the skin, subcutaneous tissues, and periosteum. The orbital rim was intact. Dissection was carried posteriorly until a defect measuring 15 by 25 mm. was exposed. The anterior margin of the bony defect was situated about six mm. within the lower orbital margin. Numerous thin fragments of bone and the lower orbital contents were displaced into the antrum. The wound was packed with gauze sponges for hemostasis while a Caldwell-Luc approach to the antrum was made through the right canine fossa. The anterior antrotomy was enlarged sufficiently to admit the examiner's finger. Degenerated fat globules floated out as the hematoma within the antrum was evacuated. Exposure of the orbital floor through the original wound, coincident with digital pressure through the lower wound, demonstrated a rent in the mucous membrane of the antral roof. An intranasal antrotomy below the inferior turbinate was performed for the purpose of subsequent drainage.

A graft of cortical bone three to four mm. in thickness was removed from the internal surface of the right ilium. The graft was shaped to fit beyond the edges of the orbital floor defect and was wired into position through drill holes in the graft and in the lower orbital margin. The orbicularis muscle and subcutaneous tissues were closed with 6-0 chromic catgut sutures. The skin was closed with 5-0 silk sutures. The Caldwell-Luc incision was closed with 4-0 chromic catgut. The right antrum was packed through the antrotomy with vaseline packing. An interpalpebral suture was placed to prevent opening of the right eye beneath a moderate pressure dressing. The wound over the ilium was closed in layers and a small rubber drain inserted.

The postoperative course was uneventful. The pressure dressing was removed in four days, the

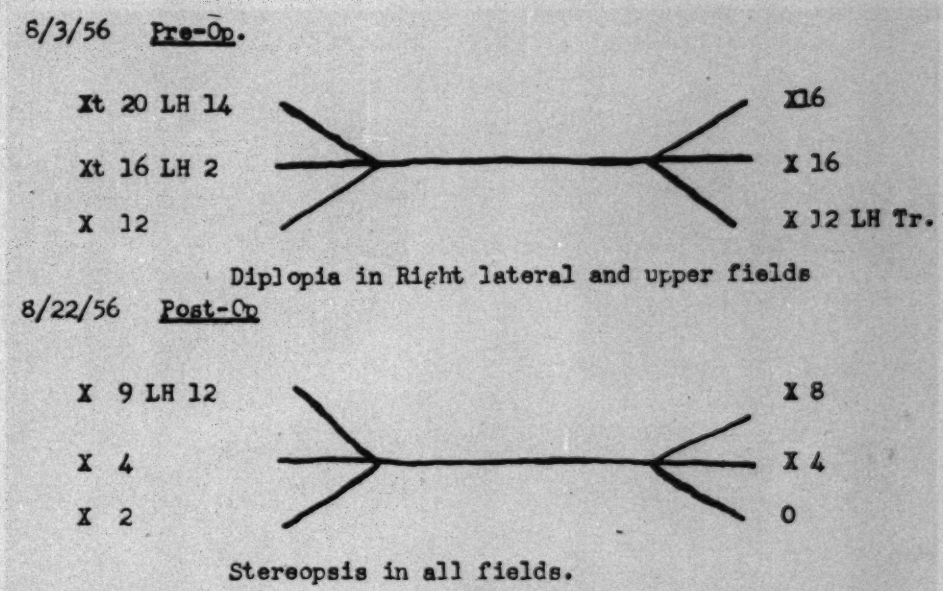


Fig. 2 (Smith and Regan). Cardinal fields preoperatively and postoperatively.

antral packing on the eighth postoperative day.

An X-ray film taken on September 7, 1956, showed the bone graft in its proper position. Residual clouding of the right antrum was present (fig. 1).

Diplopia had disappeared at the first postoperative dressing. Exophthalmometric measurements were recorded as equal on both sides.

Muscle balance at discharge on August 22, 1956, showed a small exophoria in all fields, a left hyperphoria up and right with stereopsis in all fields (fig. 2). Follow-up visits revealed no hyperphoria in any field of gaze and no evidence of enophthalmos or change in the width of the palpebral fissure. These findings were unchanged as of October 2, 1956. Postoperative X-ray films (fig. 3) demonstrate restoration at the level of the orbital floor. The position of the thin bone graft in the orbit may be seen.

Methods and results of experiment

A blow-out fracture of the orbital floor was produced in a human cadaver by duplicating a force similar to the one observed in the preceding case history. The hypotony of the cadaver globe was increased to normal finger tension by intravitreous injection of 1.0 ml. of normal saline. A hurling ball was placed over the closed lids of the cadaver orbit and the ball was struck sharply with a hammer. A cracking sound was interpreted as fracturing bone. An exploratory incision through the skin at the infraorbital margin and blunt dissection exposed a depressed comminuted fracture of the floor of the orbit. Exenteration of the orbit revealed the fracture in its entirety (fig. 4). There was also a comminuted undisplaced fracture involving the lamina of the ethmoid bone. No fracture of the orbital rim or zygomatic arch was observed. This duplicated almost exactly the injury sustained by our patient.

In a second experiment, the opposite orbit of the cadaver was exenterated. The soft tissue of the rim was excised to allow direct contact between the bony orbital circumference and the surface of the hurling ball. Repeated blows of similar force with the hammer failed to fracture the floor or the rim of the orbit. When the striking force was sufficiently increased, the orbital floor and the orbital rim collapsed simultaneously. This strengthened our belief that these fractures are caused by increased intraorbital pressure, rather than bone transmission.



Fig. 3 (Smith and Regan). Postoperative Waters view with autogenous iliac bone graft in place. Antral clouding may persist for several weeks.

Comment

The contour and thin dimensions of the orbital floor and lamina papyracea in contact with air spaces invite fracture.

The orbital floor, for nearly its entire extent, roofs the maxillary sinus with bone of from 0.5 to 1.0 mm. in thickness. This bone may be even thinner at the inferior orbital groove and canal. Behind the globe the orbital floor bulges upward presenting a thin convex surface which receives the brunt of the force transmitted by the eye. Once fracture occurs, gravity alone causes displacement of the orbital floor and orbital contents into the maxillary sinus. The amount of displacement is dependent upon the size of the antrum and the extent of the fracture (fig. 5).

Diagnosis and Treatment

Clinically, "blow-out" fractures may show laceration and abrasion of the lids with ecchymosis and edema. Chemosis and subconjunctival hemorrhage occur promptly. Emphysema is the surest indication of fracture into the nasal sinuses. Commonly, rupture of the sphincter, iritis, hyphema, commotio retinae, and transient secondary glaucoma are seen.

In fracture of the orbital floor, with or without fracture of the orbital rim, as soon as vision in the injured eye is restored, diplopia is observed by the patient, provided that binocular single vision existed prior to injury. If the natural ostium of the antrum is present, blood in the nasal cavity or postnasal discharge may be observed and nasopharyngoscopy may confirm the source. Enophthalmos and narrowing of the palpebral fissure are noticeable when orbital edema subsides. At this time orbital fat degeneration and orbital fibrosis have advanced. Dur-

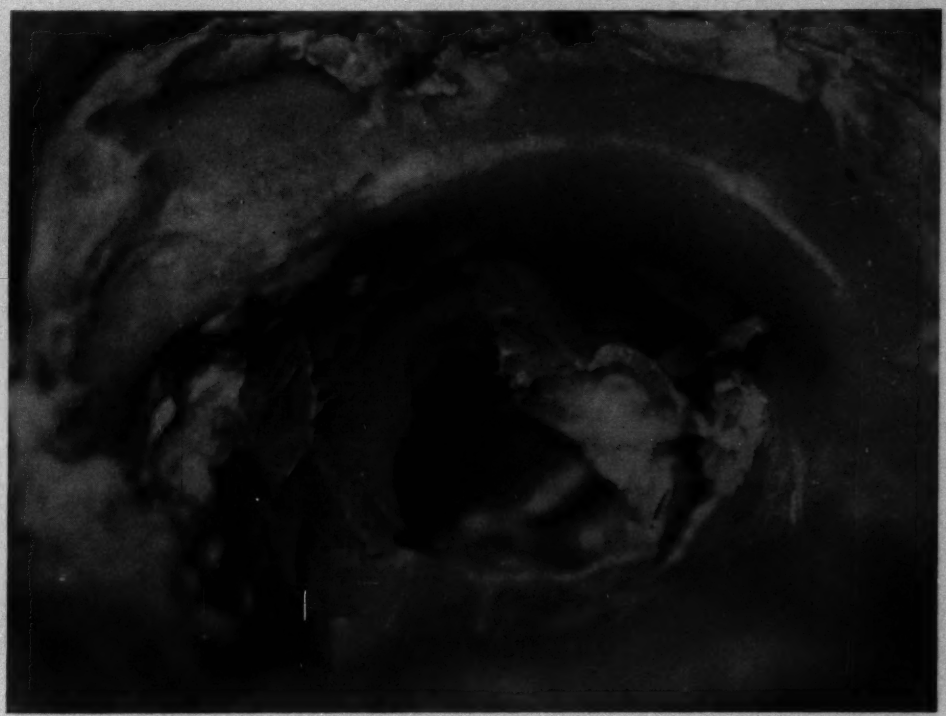


Fig. 4 (Smith and Regan). Photograph of the experimental "blow-out" fracture of the orbital floor with the orbital rim intact and fragments of the orbital floor depressed into the antrum.

ing this stage diplopia may be reduced or disappear.

Oculorotary excursions, by screen and comitance studies, are variable. Usually a restriction of depression occurs, as well as elevation of the injured eye. According to our findings and interpretations this pattern is due to herniation and incarceration of the inferior rectus and the inferior oblique muscle and their surrounding tissues into the fracture.

Absence of elasticity in the injured, impounded inferior rectus restricts rotation in the field of action of its antagonist, the superior rectus. Since the point of greatest damage is closer to the origin of the inferior oblique than that of the inferior rectus, less evidence of underaction is demonstrable in inferior oblique function.

X-ray films projected in the various orbital and sinus views characteristically show dense clouding of the homolateral antrum. Fragments of the thin orbital floor may or may not be recognized in the radiographic shadows. Especially valuable are the Caldwell and Waters views of the sinuses. Laminography is of little value in demonstrating shadows of the small thin fragments. The orbital rim and zygomatic arch show no evidence of fracture. In other words, the X-ray film may show nothing more than cloudiness of the antrum or ethmoid sinuses or both. If the X-ray films do not demonstrate a fracture in the presence of this symptom complex, it is advisable to repeat the films. Absence of X-ray findings of a specifically demonstrable fracture, however, *do not* preclude the presence of a "blow-out" fracture when antral clouding is shown.

In the presence of characteristic clinical manifestations of "blow-out" fracture of the orbit we advocate surgical exploration.

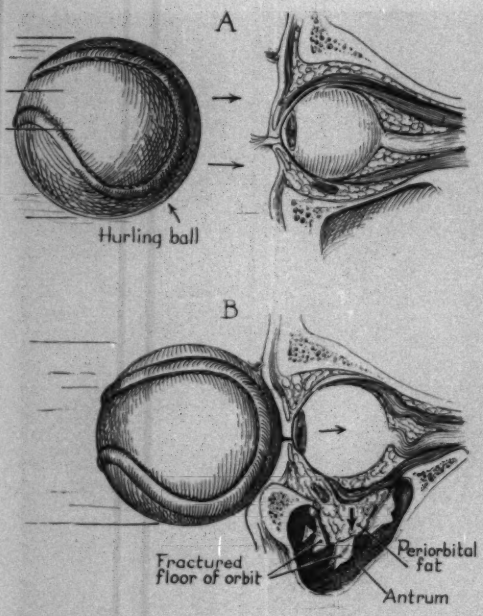


Fig. 5 (Smith and Regan). (A) The normal relation of the orbit is shown. (B) External pressure forcing a blow-out fracture of the orbital floor with incarceration of the inferior oblique and inferior rectus.

The time for exploration may be delayed as much as a week. Beyond seven to 10 days irreversible degeneration and fixation of tissues diminish the probabilities of maximal surgical restoration.

Under general anesthesia a traction test of the inferior rectus may show restriction of passive ocular elevation. The exploratory incision may be made by either of two methods: (1) orbital; (2) oral or transsinus.

Orbital exposure is made by incising the skin and underlying tissues superficial and parallel to the middle one third of the lower orbital rim. The periosteum is then incised and elevated to expose the orbital floor. A nasal speculum introduced into the incision has been found of value in exposing the depths of the wound. If the ocular exposure is used and a fracture is not revealed, no harm is done and completed healing leaves little or no scar along natural lid folds.

If the surgeon should decide upon the more radical oral approach, the typical Caldwell-Luc anterior antrotomy is done through the canine fossa. The osteotomy is enlarged sufficiently to admit the palpating finger of the surgeon. By expert instrumentation the antral roof may also be viewed through this approach. Before closure of this wound it is advisable to do an intranasal antrotomy as a precautionary measure.

In the treatment of early orbital fractures we have been disappointed with some of the results obtained from reduction and support by antral packing. We have come to believe that some type of permanent orbital floor fixation is superior to reliance on the temporary support attainable by antral packing. Of the numerous devices and materials available we are inclined to conclude that autogenous living tissue is superior to other materials. Since bone becomes consolidated and cartilage does not, our preference is autogenous iliac bone. To replace the comminuted orbital floor, a thin plate of cortical bone taken from the inner aspect of the iliac crest has proved most satisfactory. The small, separated comminuted fragments of orbital floor within the antrum should be removed. Small fragments attached to orbital tissues should be elevated and placed in contact with the upper aspect of the graft. The graft itself may be wedged into the fracture site. If it is then still mobile, a wire fixation suture may be placed in the graft and orbital rim.

Discussion

Small, blunt objects of a diameter less than that of the bony orbit tend to rupture the globe, leaving the floor of the orbit intact. Driven golf balls were the cause of rupture of the globe in three cases seen at Manhattan Eye, Ear, and Throat Hospital. We believe that objects with a diameter larger than that of the bony orbit, such as a baseball, fail to rupture the globe because part of the force is absorbed by the orbital rim. While the force is not great enough to fracture the heavy orbital rim, the soft tissues pressed into the orbital cavity penetrate

the fragile orbital floor. The fluid malleability of the contents of the globe helps prevent rupture of the globe.

Our investigation of blunt injury to the orbit leads us to believe that internal fracture of the orbit is more frequent than is commonly suspected, probably because the intact zygomatic contour tends to exclude the diagnosis of fracture. Orbital floor fracture may be observed in the absence of gross local signs although supraorbital and infraorbital edema and ecchymosis with Berlin's edema are usually seen. The initial oculorotary signs may be obscured by swelling. The posttraumatic X-ray findings of antral clouding lead one to suspect the presence of "blow-out" fracture. If definite diagnosis of internal fracture can be made, immediate surgery in the form of autogenous iliac bone graft to the orbital floor is indicated. In doubtful cases the orbital floor should be explored through a skin incision at the inferior orbital rim.

Several cases of late enophthalmos, which in retrospect would seem to be due to "blow-out" fractures, have been seen in this clinic. These late cases are far more difficult to correct.

Summary

A discussion of internal orbital fractures produced by hydraulic action of the orbital contents is presented. Experimental evidence for the mechanism of such fractures is described. A surgical means of correction and restoration by immediate autogenous bone grafting is outlined. Difficulty in early diagnosis is emphasized. Early surgical treatment tends to prevent later permanent deformity. An illustrative case is presented.

210 East 64th Street (21).

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A SIMPLIFIED METHOD FOR LOCALIZATION OF INTRAOCULAR AND INTRAORBITAL FOREIGN BODIES*

Wolfgang A. Lieb, M.D., and Herbert Wiesinger, M.D.

Richmond, Virginia

Numerous methods for the localization of intraocular foreign bodies are known. Among those the direct methods of Comberg[1] and Pfeiffer,[2] the geometric construction method of Sweet,[3] and the bone-free method of Vogt[4] are the most accurate and reliable ones.

For the practicing ophthalmologist, these have the disadvantage of necessitating specialized equipment and trained personnel. The objective of this paper is to acquaint the ophthalmologist with a simple and fast method of localization which does not require any specialized equipment but yields fair accuracy and dependability.

Method

Posteroanterior and lateral skull X-ray films are required. Any modern table X-ray

* From the Department of Ophthalmology (Chairman; Dr. DuPont Guerry, III), Medical College of Virginia.

unit may be used. In order to standardize the error due to the divergence of the beam, it is best to work with known distances. Since in most of the modern X-ray tubes the distance from the eye to the film, including the Bucki aperture, amounts to 10 cm., it is advisable to use the tube at a distance of 100 cm. This will result in a picture magnification of 10 percent. Procaine (0.5 percent) has to be instilled into the injured eye before taking exposures.

a. The *posteroanterior exposure* is taken with the patient sitting facing the X-ray film or lying in the prone position, the chin resting on a plastic sponge as demonstrated in Figure 1. The patient fixates a chalk mark on the table either with the injured eye or, if fixation cannot be obtained, with the fellow eye. In the latter case, the chalk mark should be placed slightly to the nasal side of the fixating eye in order to compensate for the convergence of the injured eye. Both position of the head and fixation are important in minimizing the deviation of the visual axis from the central X-ray beam. The blunt ends of a compass are then adjusted to the individual width of the cornea of the injured eye and held in position at the 12- and 6-o'clock meridians during the exposure (fig. 2).

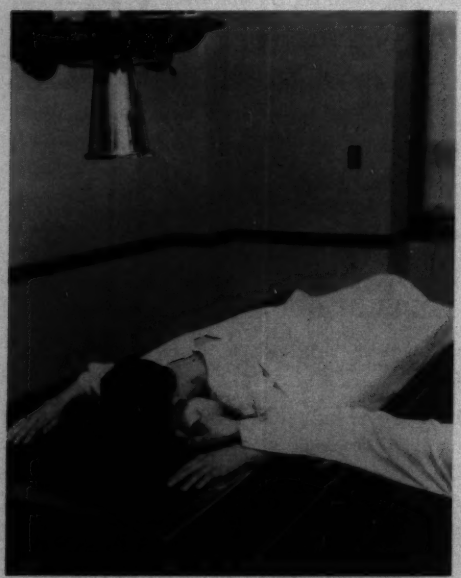


Fig. 1 (Lieb and Wiesinger). Posteroanterior position of patient for foreign body localization. The chin is resting on foam rubber. The blunt tips of the compass are held in position at the limbus.

b. The *lateral exposure* is taken with the patient either in the sitting or the lying position. The film-eye-tube distances are the same as previously discussed (10 cm., 100 cm.). The patient must fixate a mark in the primary position of gaze. The central beam should be adjusted so as to pass through the limbus, horizontal to the visual axis (fig. 3). The two compass tips are again placed at the 6- and 12-o'clock meridians during the exposure (fig. 4).

Localization

A standard 24-mm. diameter eye is assumed, and a localization scheme (fig. 5) similar to the ones introduced by Comberg[1] and Pfeiffer[2] can be used. A 10-percent correction is applied to all measurements in order to correct the magnification factor.

a. In the *posteroanterior picture*, as outlined in Figure 2, the tips of the compass are connected through a straight line (A). A second line (B) is then drawn horizontally and at right angles through the halfway point of the first line. If this line is carried across both orbits, the vertical distance from the zygomatico-frontal suture to line B should be about equal on both sides. The foreign body is then connected with the intersection of the previous lines A and B. This intersection also represents the anteroposterior axis of the eye. Thus the distance from the foreign body to the intersection (radius) and the angle between line A and radius can be carried over to the frontal plane of the scheme, after applying the 10-percent correction. A horizontal line is then drawn from the plane through the foreign body parallel to line B and through line A. The lateral distance of the foreign body from the



Fig. 2 (Lieb and Wiesinger). Posteroanterior X-ray film with compass tips in place. Arrow points at foreign body. (A), (B), and (r) represent localization lines.

sagittal plane of the eye is thus determined. This, together with the depth localization of the foreign body, is needed for the meridional scheme.

b. In the *lateral picture,* as outlined in Figure 4, the tips of the compass are again connected (line A^1). This represents the limbus plane. A line B^1 perpendicular to line A^1 is drawn through the foreign body. The distance from the foreign body to line A^1 represents the depth localization of the foreign body and this, together with the lateral distance from the anteroposterior film (fig. 2), can be carried over to the meridional scheme after applying the correction factor of 0.9.



Fig. 3 (Lieb and Wiesinger). Position of the patient for the lateral exposure.

Discussion

There are advantages and disadvantages to every known method of intraocular foreign body localization. The geometric method of Sweet introduced in 1898 and its various modifications (Stephenson[5]) is still the most commonly used one in this country. Its advantages are apparent. The localizing apparatus does not touch the eyeball which makes it especially suitable for badly lacerated globes and for use in children. The fact that it is virtually impossible to superimpose the image of the localization marker upon that of the foreign body is also in favor of this method. The disadvantage of using a standard scheme with a 24-mm. eyeball applies to the Sweet method as well as most of the others (Comberg, Pfeiffer). The error of measurement in Sweet's method becomes greater the larger the distance from the foreign body to the localizing apparatus.

This difficulty is eliminated in the direct methods of Comberg and Pfeiffer which employ a contact lens with four lead markers at the limbus. While this allows more precise measurements of the foreign body in relation to the marking points, it has the disadvantage of being unsuitable in badly lacerated globes, and that of possible superimposition of the foreign body onto the lead marks of the contact lens. The availability of only one size Comberg lens may sometimes reduce its usefulness. The bone-free methods of Vogt, Franceschetti,[6] and Larsson[7] as



Fig. 4 (Lieb and Wiesinger). Lateral X-ray film with arrow pointing at foreign body. (A') and (B') represent localization lines.

well as stereoradiography of foreign bodies (Hasselwander[8]) are an excellent adjunct for localizing small, hard to visualize foreign bodies. They do, however, require excellent patient co-operation and more specialized equipment and personnel than usual.

The method described in this paper is a simple direct method to eliminate some of the difficulties of the Comberg method. A similar approach also utilizing a compass was ap-

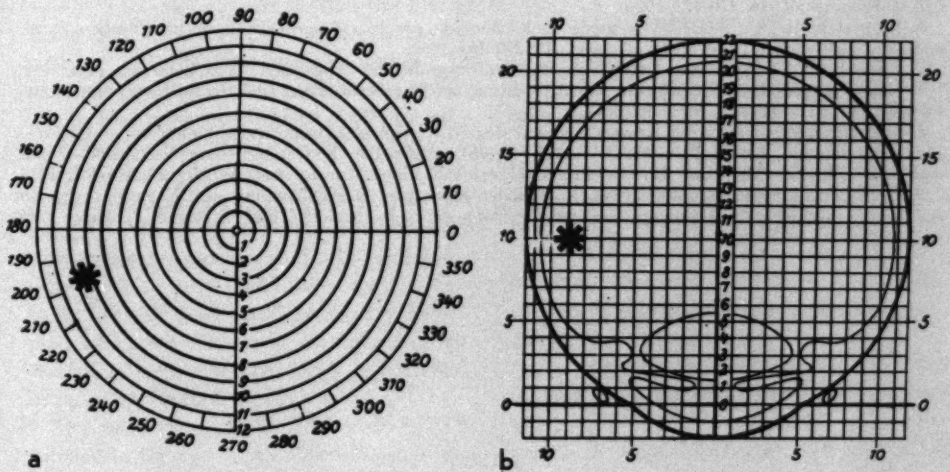


Fig. 5 (Lieb and Wiesinger). Standard 24-mm. eye scheme. (a) Position of foreign body in frontal plane. (b) Position of foreign body in meridional plane.

plied by Rohrschneider[9] at his clinic at Koenigsberg.

While all methods discussed previously are probably somewhat more accurate, the procedure we applied, in our opinion, yields dependable enough results to justify its use whenever specialized equipment is not available. Only a standard X-ray unit and a compass are necessary. The possibility of using it in badly injured globes even gives this method an advantage.

Summary

A simplified method for X-ray localization of intraocular and intraorbital foreign bodies has been described. A vertical line connecting the tips of a blunt end compass held against the cornea at the limbus is the basis for the localization. The method can safely be applied even to badly lacerated globes. The advantages or disadvantages of the various other methods are discussed.

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SURGICAL APPROACH TO INTRAOCULAR FOREIGN BODIES

MAURICE CROLL, M.D., AND LEO J. CROLL, M.D.
Detroit, Michigan

Foreign bodies within the globe can be extracted by the anterior or the posterior approach. The method of approach and the preoperative planning will determine the eventual outcome. It is not the purpose of this paper to compare the merits of the anterior versus the posterior approach. Much has been written on the subject and each method has its firm adherents.

During the Second World War, the military type of injury was more severe than any encountered in civilian practice. As a result it became necessary to work out an exact technique for removal of these foreign bodies, stressing a procedure which would produce minimal surgical trauma.

In these severely injured eyes the upper limit of physical insult the eye could withstand was unknown. The incident trauma had been suffered when the patient was first seen but the added surgical trauma was entirely within our hands. Emphasized, therefore, were (a) minimal manipulation of the injured eyes and (b) pinpoint localization and precise mm. surgery so that the foreign body could be removed with the first application of the magnet without entering the vitreous chamber. The problem was strictly one in engineering of injured ocular tissue.

With this background in mind, a plan of management slowly evolved, a step-by-step procedure, measured, controlled, and pinpointed to the exact area. It was found necessary to discard old accepted ideas and devise new ones to meet the needs until a final basic over-all plan could be worked out—a plan which was elastic in its scope and successful in removing all types of magnetic intraocular foreign bodies.

The estimation of the degree of incident damage to the eye is one that comes of long experience tempered with caution because of the many individual factors involved. As in the law of mass action, when there is an increase in one of the components the other must of necessity decrease in order to keep the total a constant. Similarly with the eye: as the degree of initial trauma is increased the amount of surgical trauma that the eye can successfully tolerate becomes less. In the anterior segment of the eye the damage to the globe can be directly visualized and estimation here is not too difficult; however, in posterior segment injuries it is not always possible to see the foreign body nor the extent of injury to the ocular structures. As a result, in posterior segment injuries one often must try to extract a foreign body in a blind pouch which is, so to speak, roughly an inch square.

In order to determine exactly what happens to the tissues of the eye when a metallic foreign body enters and has to be removed by a magnet, a series of experiments were set up to observe by direct visualization how the metallic foreign body behaves and how the vitreous, choroid, and retina react while it is being moved about by the magnet.

EXPERIMENTAL WORK

Two groups of eight experiments each, using freshly prepared pigs' eyes, were set up as follows:

The tops of the eyes were cut off about two mm. above the cornea with an exceedingly sharp knife. The purpose of this maneuver was to have the entire interior of the eye under direct view from above. Any eye in which the retina or choroid was detached or disturbed during the preparation was discarded.

The pig's eye is well suited for this type of experimental work since the structures with which we are concerned closely resemble those of the human. The pig's retina and choroid appear to be of about the same thickness and consistency as the human. The vitreous also appears to have the same gross

characteristics. However, a pig's eye preserved in formalin still is not a human living eye.

Magnetic foreign bodies of various sizes and shapes, from 0.5 mm. to 4.0 mm. in length, were inserted into the eyes at known measured distances from the site of the incision. The first group of eight experiments were carried out with a portable Lancaster hand magnet, the second group was similar in all respects except the magnet used was specially designed. This magnet will be referred to as the experimental magnet. All the experimental work was filmed by moving picture camera using a Zoomar lens and these films were studied over and over again in great detail and at different speeds.

From the experiments performed the following conclusions were drawn:

1. For an arbitrarily chosen "small" particle (1.0-mm. diameter), the portable hand magnet was not effective beyond 10 mm. from its tip. The experimental magnet was definitely effective up to 14 mm. for an equivalent particle. Beyond 14 mm. the foreign body could not be budged at all. This represents a four-mm. increase of magnetic force, that is, an improvement factor of 140 percent. This is not, however, deemed sufficient and more work is now going on in an effort to increase the power of the magnet to an over-all 18 mm., without a corresponding increase in size.

2. The sclera was dissected out in layers until the bluish color of the choroid was visible. Care was taken never to injure nor perforate the choroid. Later in the experiments a scleral button was removed, again with no damage to the choroid.

3. Every time the magnet was turned on within a 10-mm. radius of the foreign body, the particle turned on its long axis so that it always presented point first, thus making it easy to deliver. Under these conditions the foreign body acted as its own perforator. Occasionally, if the choroid bulged, it was found necessary to make a small nick in it in order to deliver the foreign body.

4. The vitreous was never cut into nor were any instruments inserted into the vitreous chamber.

5. There was a definite groove in the vitreous which followed the path of the foreign body. In some of the experiments a portion of swagged needle with a suture attached was used so that the entire path of the foreign body could be followed. Later the needle and suture were stained with fluorescein to make visualization easier.

6. In cases in which the foreign body was not delivered by the initial application of the magnet it always returned to its original position. In other words, nothing had been gained except that the vitreous had been traumatized in two directions, namely, the foreign body's path toward the magnet and its return toward its original position.

7. Whenever the magnet was turned on, the vitreous, acting as an electrolyte, was jarred in all directions throughout its entire extent, regardless of where the foreign body was located. The impression was that the vitreous was markedly disturbed with each application of the magnet. The movies of these experiments demonstrated this well.

These experiments corroborate the work of G. Strubel and L. J. Croll (Am. J. Ophth., **29**: 151 [Feb.] 1946). Their diagram (fig. 10) divides the globe graphically into regions designated as certain, critical, and failure zones according to the position of the foreign body. Regional division is essential to removal of intraocular foreign bodies.

Surgery of intraocular foreign bodies

The operation is performed under local anesthesia using a 0.5-percent Xylocaine retrobulbar injection and seventh nerve akinesia. A nonmagnetic speculum or lid suture is used.

The cornea is marked at the three, six, nine, and 12-o'clock meridians with a sharp knife dipped in methylene blue (fig. 1). Only a small nick should be made so that the dye will not run over the rest of the eye. The

dye outlines orientation marks for the operative procedure. The area of surgery is injected under the conjunctiva which is ballooned up (fig. 2). A suture (black silk 4-0) is inserted in the area, making sure that a good bite of sclera is included (fig. 3).

The conjunctiva is then incised widely for a distance of 10 to 12 mm. (fig. 4). Adequate exposure is essential. The exposed area of the sclera is scrupulously cleaned of all of its fascial attachments and all bleeding points are cauterized. The line of incision is then cauterized with a heated muscle hook to insure against infection.

The exact area of incision is outlined eight mm. from the limbus (fig. 5) in a relatively avascular area of the retina. This lessens the chance of bleeding and further injury to the eye. One end of the marker may be dipped in methylene blue to make a more vivid outline but care should be taken not to smear the entire area. The incision is made concentric to the limbus.

The scleral incision is then made (fig. 6), using long sweeps of the knife through the consecutive layers of the sclera. The pull of the scleral fibers is at right angles to the incision and, as the incision is made, they will spread apart and actually divide into consecutive layers. Figure 7 shows completion of the incision.

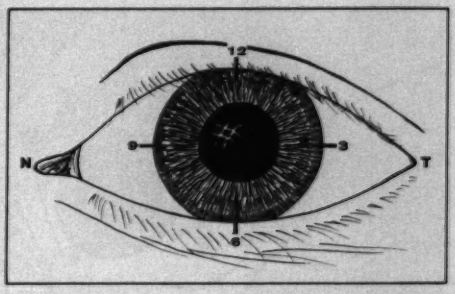


Fig. 1 (Croll and Croll). The cornea has been marked at the three, six, nine, and 12-o'clock positions with methylene blue to keep the anatomic landmarks in view at all times.

The live magnet is then brought directly to the exposed choroid (fig. 8). It is unwise to bring the magnet against the uncut sclera, for, if it attracts the foreign body, the maneuver will cause undue trauma. The choroid is never traumatized by the magnet, nor should it be caused to bleed. The choroid will give somewhat and can be indented slightly inward but it should not be stretched beyond its normal elasticity. Figures 9 and 10 show

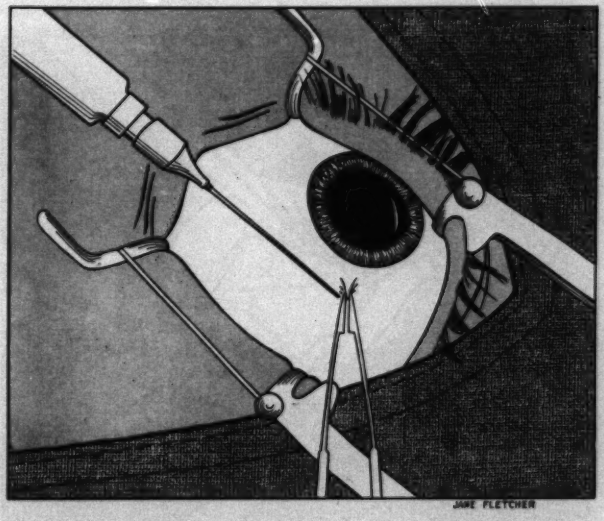


Fig. 2* (Croll and Croll). The area of surgery is injected under the conjunctiva which balloons up.

* Figures 2 through 13 are drawings based on the moving pictures taken during the procedure. We were persistent in our cinematography in order to record the exact second when the foreign body was delivered (fig. 10). Actually we had to make three separate movies before we were able to do this.

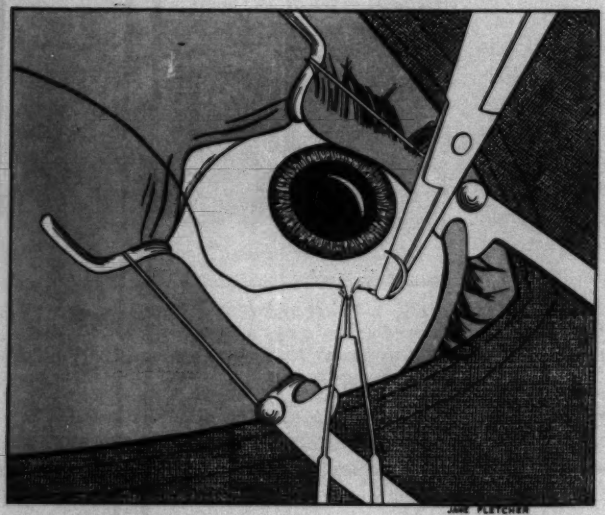


Fig. 3 (Croll and Croll). A retraction suture is inserted in the conjunctiva and upper layers of the sclera.

the foreign body coming out of the vitreous chamber, adhering to the live magnet.

The scleral incision is then closed immediately to restore the contour of the eye (fig. 11). The edges of the scleral wound are then cauterized (fig. 12) with the diathermy or with a muscle hook heated in an alcohol flame. This insures against the possibility of retinal detachment just beyond the pars plana. The conjunctiva is then tightly closed with a continuous suture (fig. 13).

Enucleated eyes

The surgical technique herein presented stresses the posterior approach through the pars plana. The anterior approach is reserved for those cases in which the foreign bodies can be seen or are present in the lens. The technique was developed by studying 101 cases carefully recorded overseas. Of these

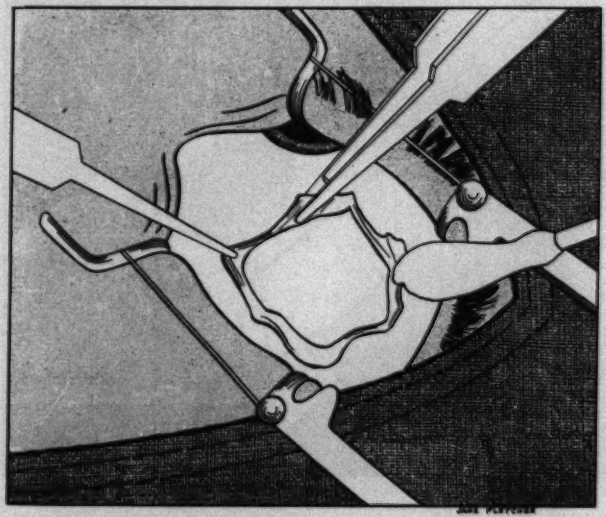


Fig. 4 (Croll and Croll). After the conjunctiva is widely incised, the sclera is bared and cleaned.

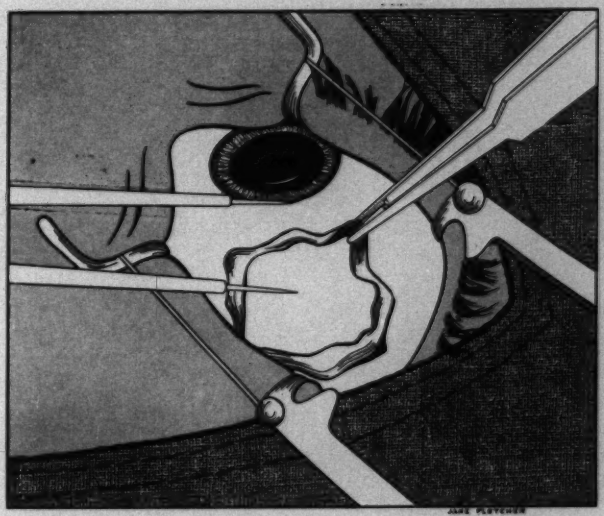


Fig. 5 (Croll and Croll). The area of incision is outlined eight mm. from the limbus in the area of the avascular pars plana.

cases, enucleation was performed in 40, the causative agent in each case being a metallic foreign body or bodies.

In reviewing the cases, an attempt was made to determine if there was a type of injury common to all the eyes enucleated. Was it possible to predict with some degree of accuracy by clinical examination the upper limit of incident trauma that an eye could withstand and still retain its viability?

The combat type of injury, violent in nature, differs in all aspects from the civilian type metallic intraocular injury. Ideally, it would have been preferable to have taken daily, detailed progress notes of each case as it developed.

All the injuries occurred overseas and certain facts regarding them should be kept constantly in mind:

They occurred mostly under combat con-

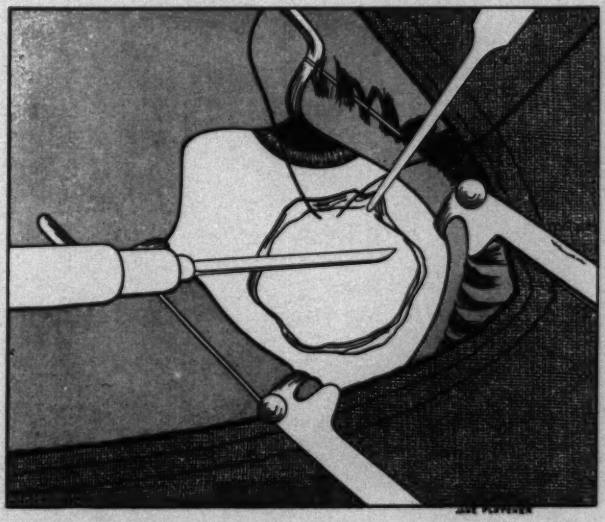


Fig. 6 (Croll and Croll). The initial incision into the sclera.

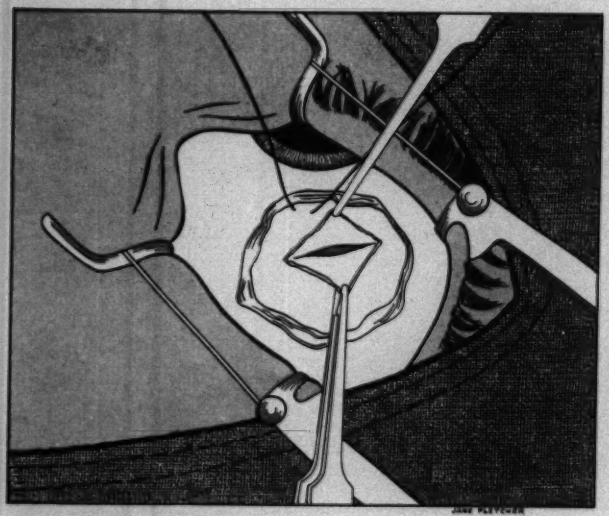


Fig. 7 (Croll and Croll). The incision has been completed and the blue of the choroid is showing through.

ditions. Many of the patients had other wounds which needed primary attention. The chain of evacuation was quite rapid and some of the patients were seen in the Zone of the Interior (United States) within 48 hours after they were wounded; 22 were in the European theater, 16 in the Pacific area; and two for whom the exact overseas area was not recorded. The average mean days from time of injury to day of enucleation was 77.40, the longest being 188 days, the shortest 25. When first examined 16 eyes were totally blind, 13 had only light perception, three had light perception and projection. In five cases there was no vision recorded and in the remaining three, the vision was 20/70, 3/200, and 20/200.

The predominant feature in all the eyes was the presence of hemorrhage into the vitreous, probably from the choroidal bed

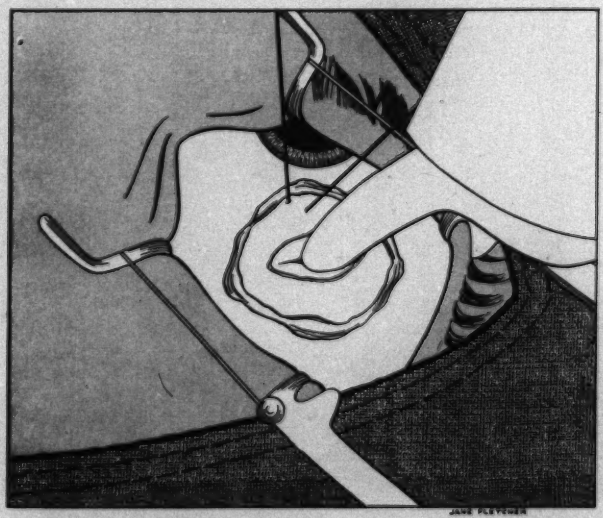


Fig. 8 (Croll and Croll). The live magnet is shown in direct apposition to the choroid.

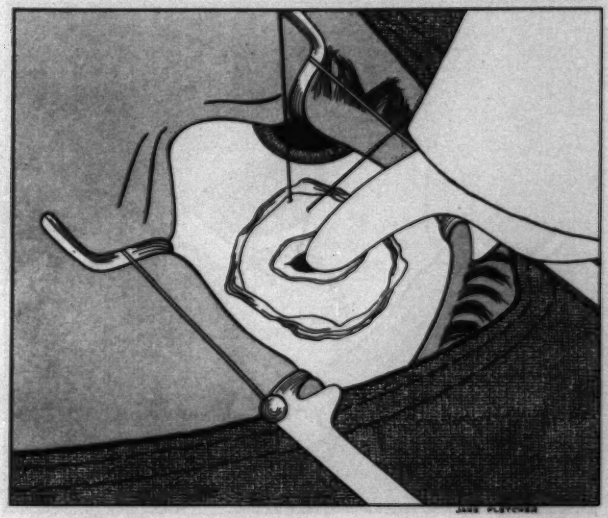


Fig. 9 (Croll and Croll). Actual delivery of the foreign body as it is coming out of the vitreous chamber.

and the retinal vessels. Damage to the vitreous as a result of hemorrhage varied in amount and degree but in all cases it proved to be overwhelming. Since the vitreous is approximately two thirds of the volume of the globe and has a very low metabolism, the ability to repair itself is not very great. It does not withstand traumatic insult well. Added to this is the severe damage to the vascular nourishing choroid, a damage which, by itself, quite often proves fatal to the eye. Vitreous hemorrhage occurred in 24 eyes.

In addition to posterior segment injury to the vitreous, separation of the retina was also present in 11 cases. This is out of proportion to the number of vitreous hemorrhages. It must be remembered, however, that in many cases the retina could not be visualized. Due to massive intraocular hemorrhage, many of the eyes did not give any red reflex and it

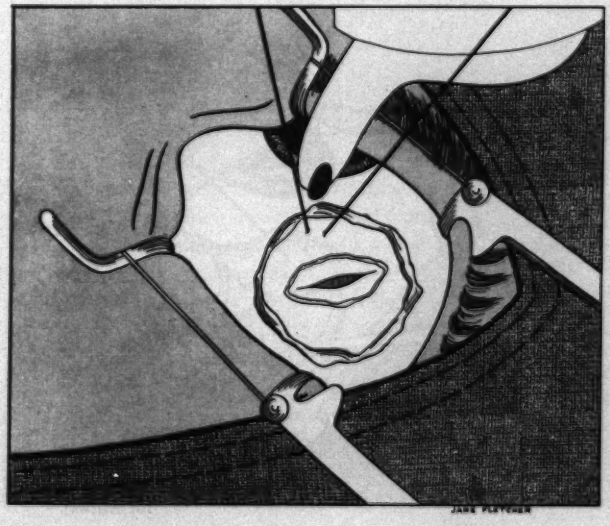


Fig. 10 (Croll and Croll). The foreign body after it was delivered.

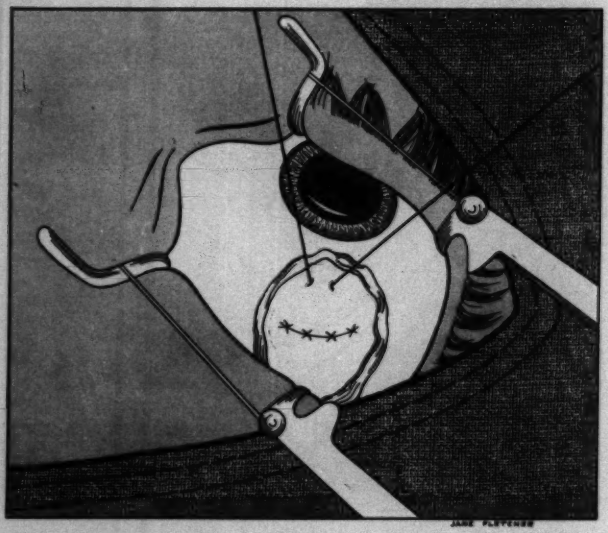


Fig. 11 (Croll and Croll). The sclera is closed immediately.

was, therefore, impossible to determine the presence of retinal detachment. Although there may have been a larger number of retinal detachments, we can only list the 11 detachments observed.

Another type of injury that was frequently seen in this series was a traumatic uveitis not at all like the types commonly seen in every-day ophthalmic practice. The infection proved to be intractible and did not respond to any of the known antibiotics. It had a persistent, gradually progressive, down-hill course, and eventually led to destruction of the eye. This type of chronic, severe infection occurs in traumatized eyes and is due to the various types of debris and foreign material carried into the eye at the time of injury. These findings were confined in most cases to the anterior segment of the eye.

No etiologic organism could be cultured

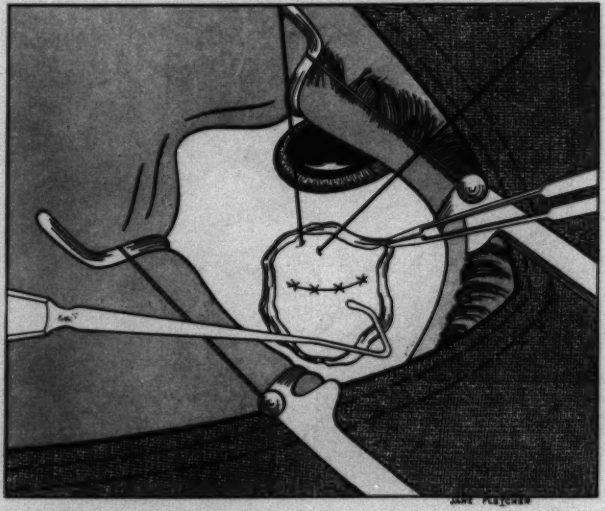


Fig. 12 (Croll and Croll). The cautery is applied to the far side of the incision to insure against the possibility of retinal detachment.

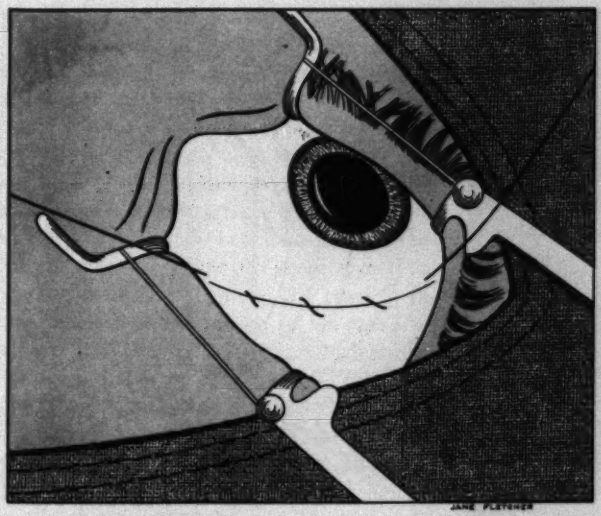


Fig. 13 (Croll and Croll). The conjunctiva is closed with a running suture.

at any time from these eyes. These injured eyes offered little resistance to the infection and rapidly disintegrated to complete atrophy. What part the phaco-anaphylactic reaction of liberated lens material and scattered uveal pigment played could not be evaluated; yet it was a contributing factor. There were 15 cases of this type of uveitis, five cases of iridocyclitis, and only two cases of secondary glaucoma. The two glaucomas occurred late in the course of the infection and hastened the eventual destruction of the eye.

Siderosis was present in seven cases. This, however, is not a true evaluation because the follow-up in some cases was too short. In other words, the eyes were enucleated for various other reasons before the chemistry of siderosis could develop. Siderosis may develop in any eye which contains an intraocular foreign body or in any one from which a foreign body has been successfully removed. Especially is this true if the foreign body is in the ciliary body, iris, or posterior chamber. In five of the cases, a severe uveitis was also present, or an iridocyclitis; the remaining two cases had iris disturbances, adherent leukoma, and posterior synechias. In all seven cases, the eyes became atrophied and it was necessary to enucleate them. It should be kept in mind that all of these injuries were accompanied by other injuries.

TYPES OF INJURIES

Corneal opacity	4
Iridocyclitis	5
Uveitis	15
Cataract (traumatic)	11
Vitreous hemorrhage	24
Chorioretinitis	7
Retinal detachment	11
Atrophy of globe	20
Siderosis	7
Glaucoma (secondary)	2
Sympathetic ophthalmia	1

Miscellaneous (infrequent injuries)

Dacryocystitis	1
Paralysis of ocular muscles (all in 1 case)	5
Avulsion of optic nerve	2
Enophthalmos	2
Cicatricial deformity of canthus	1
Severance of canaliculi	2
Fracture of orbital wall	2
Fracture of malar bone	1
Fracture of superior maxilla	1
Deformity of upper lid (notching)	1
Rupture of iris sphincter	1
Synechias (posterior)	1
Leukoma (adherent)	1
Coloboma of iris (traumatic)	1
*Hemorrhage into anterior chamber	1
*Hemorrhage into choroid	1
Hemorrhage into retina	1

Average mean days of enucleation after injury 77.40.

* Same case.

VISION

Blind	16
Light perception only	13
Light perception and projection	3
Not recorded	5
Miscellaneous 20/200, 20/70, 3/200	3

In summary, then, 40 eyes were enucleated: 32 had retained foreign bodies. In the remaining eight, the initial injury was caused by foreign bodies, although there were none present at the time of operation. There was only one case of sympathetic ophthalmia caused by a foreign body (6 by 3 by 2 mm. in size) and the exciting eye was enucleated 37 days after injury.

In 31 cases the major portion of the pathologic alteration was located in the posterior segment of the eye, that is, behind the lens. In two cases the injury was confined largely to the anterior segment, that is, anterior to the lens. In two cases no major area could be accurately designated. In five cases the records were not definite enough to designate whether the injury was in the anterior or posterior segment.

MAGNETS AND MAGNETISM*

Two types of magnets are commonly used in ophthalmology: (1) the giant and (2) the portable hand electromagnet. The giant magnet is a large electromagnet which, because of its great size and weight, is cumbersome and unwieldly. It does not lend itself to the precision techniques discussed in this paper. The hand-type electromagnet, illustrated in Figure 14, is a straight core of soft iron, with many turns of fine wire around this core. Various shapes of magnet tips are available. A particular tip is attached to the magnet core by threading it into a tapped hole at one end of the core.

Although this magnet has been successfully used in many studies, its use is limited by the inefficiency of its magnetic design. The experimental magnet used for some of the studies discussed in this paper is an attempt to overcome this deficiency in design by simple modification of the existing magnet. The effectiveness of this modification may be readily observed by noting that the 10-mm. range of the original hand electromagnet was increased to a range of 14 mm., an improvement of 140 percent. Figure 15 illustrates the modified magnet.

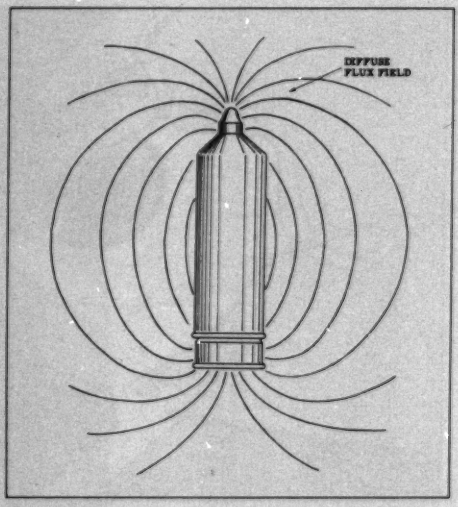


Fig. 14 (Croll and Croll). The portable hand magnet, showing the flow of the lines of flux around the magnet.

In order to explain the magnet modification, certain fundamentals of magnetism should be outlined:

Any magnetic particle in a nonuniform magnetic field will tend to move toward the strongest region of the field. If the particle is free to move it will come to rest in that position in the field where the magnetic flux lines crowd together most densely. The force which causes a restrained particle to overcome restraint and to move toward the strongest portion of the field depends upon three factors: (1) the strength of the field in the original position of the particle, (2) the rapidity with which the field strength changes near the original position of the particle, and (3) the volume of the magnetic particle. The modification which resulted in the "experimental" magnet proposed to in-

* We are indebted to R. Essling for the section on "Magnets and magnetism."

crease the first and second items.

By putting a magnetic shell around the coil of the original magnet and by forming the magnet tip so that magnetic flux was concentrated in a short gap of a small cross-section, the magnetic field strength around the tip was appreciably increased and the rate of increase of magnetic field strength as the tip is approached was also markedly increased. For a particle of given size then, that force tending to move the particle toward the flux concentration in the magnetic gap was greater than that force which tended to move a similar particle toward the blunt tip of the original hand magnet.

The reason that the magnet modification used in these studies increases the field strength and the rate of change of field is illustrated in Figures 14 and 15. First, the magnetic field or flux field around the unmodified magnet is shown in Figure 14. It may be observed that all flux lines leaving the tip of the magnet must re-enter the magnet at its opposite end. These flux lines rapidly diverge and turn back, away from the region where a high flux concentration is desired.

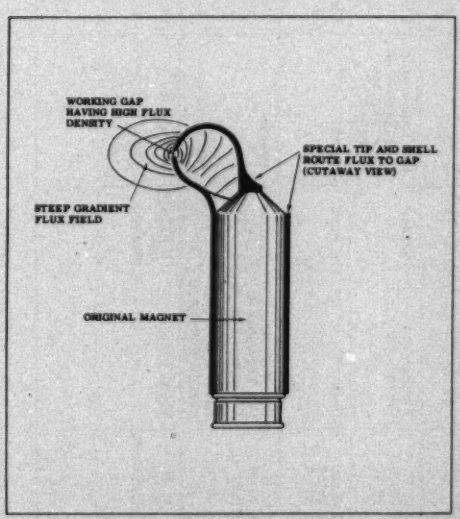


Fig. 15 (Croll and Croll). The experimental hand magnet.



Fig. 16 (Croll and Croll). X-ray film of foreign body in case of E. B. (Case 1, table 1.)

Figure 15 shows that, in the modified magnet, a soft iron shell around the magnet coil routes the flux through a path of low resistance so that high flux densities are everywhere maintained.

Because of the small cross section of the soft iron path at the working tip of the magnet, the soft iron becomes magnetically saturated near the tiny gap existing at this working tip. This causes many of the flux lines to leak around the gap by a longer route. However, since the air path taken by this leakage flux is much shorter than the air path followed by flux lines which go from one end of the unmodified magnet to its other end, a higher concentration of flux exists around this working tip of the modified magnet than exists near the tip of the unmodified magnet. Moreover, since a relatively high percentage of the total flux actually goes directly across the working gap, the greatest field strength possible with the modified magnet is greater

TABLE 1

SURVEY OF 15 CIVILIAN CASES OF INTRAOCULAR FOREIGN BODIES CHOSEN AT RANDOM

	Cornea	Anterior Chamber	Iris	Lens	Vitreous	Choroid & Retina	Intraocular Foreign Body	Surgery	Comment
Case 1. E. B. V: O.D. 20/200 O.S. 20/20 11-6-48	Clear	Normal but shallow	Normal	Normal	Slightly hazy	No detachment	8 mm. above H; 3 mm N; 22 mm. behind cornea	11-8-48 F. B. delivered 1st attempt hand magnet posterior approach	V: O.D. 20/20 O.S. 20/20 18 mo. follow-up
Case 2. G. C. V: O.D. 20/100 O.S. 20/20 12-29-48	Through and through wound	Shallow depth	F.B. imbedded in iris at 6-o'clock	Clear	Clear	Good detail No detachment	No X-ray film—F.B. visible	12-29-49 F.B. removed by anterior route 3rd attempt	V: O.D. 20/20 O.S. 20/20 4 yr. and 11 mo. follow-up 10-11-54
Case 3. M. B. V: O.D. light percep & proj. O.S. 20/50 +2	Through and through wound	Normal	Normal	Rupture	Normal	Normal	No F.B. patient picked it out herself	X-ray films shows F.B: No surgery	V: O.D. 20/50 O.S. 20/30 with correction 5-23-53
Case 4. W. C. V: O.D. 20/50 O.S. 20/20 11-22-48	Clear	Normal	Normal	Clear	Slightly hazy	No detachment	One F.B. 7 mm. below H; in center 22 mm. back of cornea	F.B. delivered 1st attempt 11-24-48	V: O.D. 20/40 O.S. 20/30 with correction 5-22-49
Case 5. K. H. No vision taken; Emergency at Hospital 1-1-48	Clear	½ full of blood	What is seen is normal	Clear above	Not seen	Cannot be seen	2 F.B. glass particles by X-ray films	2 F.B. picked out of iris anterior route	V: O.D. 20/40 O.S. 20/30 Both with correction 5-22-48
Case 6. K. B. V: O.D. 20/20 O.S. 20/40 4-21-47	Through and through scar	Shallow	F.B. imbedded in iris Triangular in shape with base buried in iris	Clear	Hazy	Details not seen well but no detachment	1 F.B. present	Delivered 5th attempt anterior route	V: O.D. 20/20 O.S. 20/20 No follow-up
Case 7. W. N. V: O.D. 20/30 O.S. 20/20 11-21-52	Clear	Normal	Normal	Clear	Hazy	Details fuzzy	1 F.B. located 2 mm. below H; 2 mm. to T; 13 mm. back of cornea	Delivered 1st application of hand magnet, pars plana posterior	V: O.D. 20/20 O.S. 20/20 12-5-52
Case 8. L. H. V: O.D. 20/20 O.S. light percep. 2-45	Opacified; struck in O.S. in combat in Philippines and immediate loss of vision;o perated overseas; no F.B. found	Shallow	Atrophic	Traumatic cataract	Not well seen	Not well seen	1 F.B. brass; operated 11-45	Enucleated unable to remove F.B.	Initial trauma too great for eye; persistent pain and iridocyclitis
Case 9. A. M. V: O.D. 20/20 O.S. Fingers at 2 ft. 12-31-47	Clear	Normal but shallow	Normal	Clear	Hazy	Retinal edema and preretinal hemorrhage	1 F.B. floating free in vitreous located 10 mm. back of cornea; 8 mm. below H; 2.5 mm. nasal	Another check localization was done and in this position patient was sandbagged and wheeled to operat. room in fixed position. F.B. removed 1st attempt hand magnet	V: O.D. 20/20 O.S. 20/70 5-12-48

	Cornea	Anterior Chamber	Iris	Lens	Vitreous	Choroid & Retina	Intraocular Foreign Body	Surgery	Comment
Case 10. S. J. V; O.D. 20/70 O.S. 20/50 10-12-50	Through and through laceration	Normal depth	Small tear at 11-o'clock	Small confined linear opacity superiorly	Clear	No hemorrhage; no detachment	1 F.B. located 7 mm. back of cornea; 1 mm. above H	F.B. delivered from lens 1st attempt hand magnet	Original cataract ous area did no spread V: O.D. 20/50, 20/30 (corrected) O.S. 20/50, 20/20 (corrected) 1-56
Case 11. V. D. V: O.D. no light percep. O.S. 20/20 12-3-47	Through and through scar	Very shallow soft eye	Chronic iridocyclitis with great deal of pigment	Traumatic cataract	Not seen	Not seen	2 localizations indicate F.B. is not fixed in on. area	2 attempts; 2nd successful 14 da. 1st attempt; hand magnet	V: O.D. light percep. & proj. O.S. 20/20 When first seen O.D. was blind; after surgery see light. 4-5-48
Case 12. T. G. Unable to get initial vision 12-27-48	Normal clear	Normal depth	Normal	Clear	Slightly hazy	Large retinal hemorrhage superior temporal area	F.B. 5 by 5 by 5 mm.; extraocular BB shot	BB reached through conjunctival incision	An object of this size if intraocular would certainly cause a great deal of destruction
Case 13. A. T. V: O.D., No light percep. O.S., Normal 11-20-53	Seen in hospital as emergency	¾ full of blood; eye soft and sunken	Large iris prolapse	Not seen	Not seen	Not seen	X-ray no F.B.	Iris prolapse repaired lens later removed (F.B. struck patient's eye and ricochetted off)	Piece of steel struck patient O.D. hard enough to knock him off his stool.
Case 14. R. D. Seen as emergency in Hospital 12-24-53	Through and through cut in cornea	Shallow soft eye	Hole in iris	Cataract confined to one area	Hazy	2 large hemorrhages	F.B. floating free in viterous	Pars Plana approach; delivered 1st attempt hand magnet	V: O.D. 20/20 O.S. 20/40 (corrected); cataract confined to initial area 2-10-55
Case 15. R. H. V: O.D. 20/20 O.S. 20/200	Through and through scar	Very shallow	Small hole in edge of iris	Traumatic lens (confined to small area when first seen)	Normal	Normal	F.B. in lens	Anterior route; removed 1st attempt hand magnet	V: O.D. 20/20 O.S. 20/30 (with contact lens) X-ray did not show F.B. until free projection made



Fig. 17 (Croll and Croll). X-ray film of foreign body in case of W. C. (Case 4, table 1.)

than the greatest field strength possible with the unmodified magnet.

Since the flux density can reach a higher value in the modified magnet than in the unmodified magnet, a greater change of flux density, or of field strength, exists near the tip of the modified magnet. It is the improvement in these two factors—(1) the field strength around the working tip and (2) the rate of change of that field strength—which causes a greater pull to be exerted on a magnetic particle in the magnetic field.

A complete redesign of the magnet is now being studied in hope that a range of at least 18 mm. may be reached. With a magnet of that range, any particle 0.5 mm. in diameter or larger could be removed from any part of the eye by the techniques herein described.

Civilian cases

Out of a group of 35 cases, we chose 15 cases at random for this report (table 1). Certain basic principles were established for this study:

1. We considered two years a minimum follow-up because, after surgery, many types of complications may develop, such as siderosis, recurrent retinal detachment, and even eventual degeneration of the eye.

2. Antibiotics were used in all cases whether infection was present or not.

3. The management of intraocular foreign bodies begins the moment the diagnosis is made.

4. The surgical management begins with the preoperative planning—two Sweet localizations that coincide. This perhaps is the most important step in the entire proceedings. It pinpoints the foreign body to the exact millimeter and minimizes surgical trauma.

5. The approach used was the posterior type through the relatively avascular pars plana. If the foreign body was in the anterior segment, it was removed through a corneal incision.

6. The magnet used was the Lancaster hand-magnet or the newly devised experimental magnet. It was felt that, with pinpoint localization, it was not necessary to use the giant magnet, so it was never employed.

7. The vitreous was never entered with the tips of the magnet; the vascular choroid was never disturbed. The sclera was dissected in layers until the blue of the choroid was seen. The magnet was then applied to the choroid without breaking its continuity or drawing



Fig. 18 (Croll and Croll). X-ray film of foreign body in case of A. M. (Case 9, table 1.)

any blood. In a few instances it was necessary to nick the choroid in order to permit the foreign body to deliver. The choroid closed immediately of its own accord.

8. If the foreign body was in the lens, it was delivered first and the cataract was taken care of later. It seemed important to keep the posterior capsule intact so that the foreign body would not be lost in the vitreous, necessitating a second major procedure.

Conclusions

A technique of posterior approach for the removal of intraocular foreign bodies, based on our experience in the Second World War, has been outlined step by step. The surgical procedure, as shown by the illustrations, is an exact technique which has proved highly successful in all cases in which the initial trauma was not too great.

1326 Maccabees Building (2).

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OPHTHALMIC SURGERY IN INDIA

Richard A. Ellis, M.D.
Philadelphia, Pennsylvania

From November, 1954, to March, 1955, I worked with various missionary ophthalmologists in India and Pakistan. The practice of ophthalmology differed in many respects from that in the United States and consisted primarily in eye surgery. To appreciate fully the reasons for this difference in our specialty, one must consider certain social and economic conditions which prevail in that section of the world.

There is an acute shortage of well-trained physicians. For a population of approximately 375 million in India, there are relatively very few well qualified doctors. While 85 percent of India's people live in villages, these doctors, for obvious reasons (better educational facilities and social contacts) prefer the more lucrative practice in the larger cities.

There are also approximately 600,000 unqualified doctors or quacks. Some of these "doctors" have no training at all; others have had short inadequate courses. I met one of these local practitioners whose previous occupation was cooking at a mission hospital. He then obtained a "medical diploma" after

a one-week course. This "diploma" certified that the practitioner was well qualified to give medicine intravenously, intramuscularly, and subcutaneously. He operated a 17-bed private hospital only 100 yards from the mission hospital. His practice flourished and he even wanted to hire a well-qualified doctor to work for him. Anyone may purchase drugs including antibiotics or narcotics without a prescription.

Because of the appalling shortage and with the idea that some aid is better than none, the government does nothing to stop these unqualified practitioners. For the qualified practitioners, there are no specialty boards comparable to ours in the United States.

Despite making great strides since independence, extreme poverty is still prevalent. The average wage for those who were fortunate enough to be working in Mungeli, one of the villages where I worked in Central India, was one rupee a day; the equivalent of 21 to 22 cents. Therefore, many cannot afford modern drugs and medical treatment. Hospitalization time must be kept to a minimum. One can understand why a poor farmer, working in his fields during the yearly harvest time, would of necessity wait until after the harvest before seeking treatment for his painful inflamed red eye.

Social custom, religion, and superstition also influence the practice of ophthalmology. The government has outlawed the caste system in India but it still exists in the villages. People of one caste will not eat food prepared and served by a person belonging to another caste. Most patients entering the hospital bring their own relatives who will cook and serve them while they are hospitalized. I recall one patient who walked barefoot for three days and nights to reach the hospital. Rather than accept food from the mission during convalescence from his cataract operation, he chose to go home and later return for surgery with his relatives who would feed him. There was another patient who suddenly left the hospital on the fourth day after a cataract extraction without any explanation. The other patients explained to us that he had not eaten any food since it was not prepared by someone from his caste. Finally, when he could no longer stand the starvation, he quietly left the hospital and returned home.

Many religious Hindus do not eat meat. Cows are considered sacred in India and allowed to roam freely through the streets and houses. They certainly do not help the sanitation problem. Because they are so emaciated, they produce very little, if any, milk and only add to the economic burden of India. During my stay in India, I drank water buffalo milk.

Cow manure is shaped into 10 to 12 inch patties by hand, put in the sun to dry and then used for fuel. Cow dung is also used for a plasterlike coating on the walls and floors of the houses, many of which are made of mud. Many pious Hindus drink cow urine to sanctify themselves and some diseases are treated by drinking the urine.

Other methods of self-treatment and home remedies are: making offerings and vows to various goddesses, applying ashes to sores, ingesting no cold food or no food at all, or tying a rupee, a coin, in a cloth around the neck of the patient. Regardless of the severity of their symptoms, most of the people would not seek medical treatment during a holiday, or if it were not considered an auspicious time.

(Fig. 1-A) Vitamin-A deficiency in a 16-year-old boy, exhibiting typical Bitôt's spots. This patient also suffered from nyctalopia.
(Fig. 1-2) Close-up view of left eye of same patient, showing Bitôt's spots located on the temporal interpalpebral conjunctiva. The base of the triangular shape is at the limbus.
(Fig. 2-A) Xerosis in a 20-year-old man. The visual acuity was reduced to hand movements.
(Fig. 2-B) Close up of left eye of same patient, showing xerosis of conjunctiva and cornea. There was wrinkling of the dried out conjunctiva. Corneal sensation was also reduced.
(Fig. 3) Keratomalacia involving both eyes of an 18-month-old child.
(Fig. 4-A) Trachoma stage II, showing papillary hypertrophy (the lid is everted).
(Fig. 4-B) Pannus formation at upper limbus in trachoma.
(Fig. 4-C) More advanced stage of trachoma, exhibiting entropion, corneal scaring, and pannus formation.



Fig. 1A



Fig. 1B



Fig. 2A



Fig. 2B



Fig. 3



Fig. 4A



Fig. 4B



Fig. 4C



Fig. 4D



Fig. 4E



Fig. 4F



Fig. 4G



Fig. 5A



Fig. 5B



Fig. 5C



Fig. 6

Facilities for transportation and communication are limited. Air travel exists only between the large cities and the cost is prohibitive to the majority of people. Train and bus travel are also inadequate so that transportation is largely by ox cart and by foot. Because of this, medical care is frequently not available though it may be only 10 miles away.

The weather in India and Pakistan also influences medical practice. There are three seasons of the year, each lasting about four months. During the winter months from November to February the weather is ideal. The daytime temperatures range in the 70's, the nights being cooler. However, starting in March, the hot weather begins. The daytime temperatures reach 120° F. or more and reduce hospital activity to a standstill. Missionaries try to do their work from 5:00 A.M. to 8:00 A.M., since the heat then forces them to lie in shelter during the day. The monsoon season then follows and the constant rain causes creeks and rivers to overflow, washing out roads and stopping most travel. Thus, most hospital work is done only in the winter months. From a practical standpoint, only during one third of the year is the weather favorable for the patients to seek proper medical care.

About 90 percent of the people are illiterate. Many of them are easily misled by quack practitioners even when well-trained doctors are available. Poor education also goes hand in hand with poor sanitation. Proper sewage and toilet disposals are rare; human feces are frequently mixed with soil used for raising crops. Most water and milk are not safe to drink and there is an overabundance of filth and flies. Malnutrition and vitamin deficiency are common, even when all the essential foods may be available.

Because of these previously mentioned living conditions, the practice of ophthalmology consists mainly in eye surgery. Once a patient comes for eye treatment, it is unlikely that he will ever see a trained ophthalmologist or even a qualified physician for the rest of his life. Therefore, the most definitive form of treatment is given and the majority of patients undergo surgery. I do not wish to imply that needless surgery is done merely for the sake of doing something. No matter how conservative one's standards may be, there are far too many patients who honestly require a surgical procedure for any surgeon to consider, even remotely, unnecessary surgery.

The most frequent eye operation is cataract extraction. Preoperatively external examination includes the recording of the ocular tension with a tonometer. Ophthalmoscopic examinations are also considered routine. However, slitlamp examinations are only performed when considered necessary.

Patients in the hospital usually have an urine analysis and a blood-pressure test but no physical examination. Prior to surgery, the patients wash their faces and the nurse cuts their eyelashes. The patients would then be lined up on benches or squat on the ground outside the operating room where a nurse would perform a seventh-nerve block with two-percent Procaine and instil a local anesthetic into the eye. As a rule only children were given general anesthesia. The eyelids were also cleansed with 2.5-percent tincture of iodine followed by alcohol.

Usually three operating tables were in constant use and the surgeon would move from table to table and lose no time waiting for a patient to be prepared. When the patient was

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(Fig. 4-D) Bilateral entropion due to cicatricial stage of trachoma in a 35-year-old woman.
(Fig. 4-E) Close up of severe entropion due to trachoma in the same 35-year-old woman.
(Fig. 4-F) Postoperative result six days after plastic correction of lid for entropion due to trachoma.
(Fig. 4-G) Close up of same patient six days postoperatively. The cotton sutures are still present.
(Fig. 5-A) Leproma of cornea in a 45-year-old man.
Figs. 5-B and 5-C) Leprosy in a 50-year-old man, exhibiting loss of hair of the eyebrows, typical cutaneous changes in the lids and adjoining skin, ectropion due to seventh nerve involvement, loss of corneal sensation due to fifth nerve involvement.
(Fig. 6) A 12-year-old boy who recovered from smallpox six months earlier exhibits dense corneal scars and typical facial scarring.

on the operating table, eyes were flushed out with one-percent mercurochrome and then 1:4,000 bichloride of mercury. Anethane and 1:1,000 adrenalin were also instilled. The operating physician gave the retrobulbar block and repeated the facial nerve block if it was not satisfactory.

Gloves were not used; however, great care was taken never to allow any suture or instrument to touch fingers or lids prior to use in the eye. Each instrument was used only once and then re-sterilized. There were two or three well-trained nurses constantly sterilizing all instruments. Since there was no electricity, one nurse would direct a large flashlight on the operative site. Graefe knife sections were used in most cases, as well as three postplaced corneoscleral sutures. If surgery for both eyes was indicated, they were usually both operated upon during the same "sitting."

After surgery, the patients were either carried or led to their floor mats or beds in their quarters by an attendant. They remained there and were followed for eight to 10 days, at which time the sutures were removed and they were discharged with local medication. Of course, if there were any complications, they were kept longer. Most patients, being illiterate, were given a simple +10D. sph. lens. If any of them so requested, they were given a refraction.

Glaucoma surgery was second in frequency and, again, bilateral operations were done at the same time. Iridencleises and trephinations were most commonly performed. Because of the previously mentioned economic and social conditions, medical therapy was not feasible in most instances. Patients could not afford miotics, even if they were available, and could not be relied upon to use them daily. Therefore, the indication for surgery would consist merely of making the diagnosis of primary glaucoma.

Plastic surgery for entropion of the lids due to trachoma was next in frequency.

Dacryocystorhinostomies, optical iridectomies, and pterygium excisions were other frequent procedures. Strabismus and retinal detachment surgery was relatively uncommon.

Because of religious beliefs, the people were very reluctant to undergo loss of any part of their body. Therefore enucleations were very rare.

Besides operating in the hospital, we would also visit villages 25 to 100 miles away. At these camps, from 75 to a few hundred operations would be performed in one to two days. We operated in tents or small buildings, such as school houses, at these camps and the patients were followed for eight to 10 days.

In spite of all the handicaps, the operative results were very gratifying. I was particularly surprised by the very few postoperative infections in spite of the absence of routine systemic antibiotics. There was only one complication that never is seen in our country; postoperatively, the patients were lined up on the ground like sardines and the result was that an occasional face was stepped on.

Besides having all of the diseases that we have in the United States, India and Pakistan have many other conditions rarely, if ever, seen in our country.

Vitamin-A deficiency was not at all rare. The eyelid margins become covered with sticky secretions and the conjunctiva becomes dry and wrinkled. Many cases exhibiting Bitôt's spots were seen (figs. 1-A, 1-B, and 1-C). These spots appeared as white, sharply outlined, foamy patches on the conjunctiva. They were typically triangular in shape, with the base at the limbus, and were found in the interpalpebral opening on the temporal side.

Nyctalopia was present in these patients. As the deficiency continued, the xerosis of the conjunctiva and cornea (figs. 2-A and 2-B) increased and then developed into keratomalacia (fig. 3). In this advanced stage of deficiency, the corneas became cloudy and then necrotic. Perforation with

little inflammatory reaction frequently resulted. In other cases, secondary infection with all of its complications occurred. Needless to say, there were many cases of blindness due to this entirely preventable deficiency. At one missionary hospital where I worked, they routinely prescribed vitamins for all the surgical cases, obviously with very good reason.

Trachoma was present in about 80 percent of the patients I examined. All of the typical stages were seen (figs. 4-A to G). The active infectious cases seemed to respond to any of the following medications: Sulfadiazine, penicillin, aureomycin, terramycin, and chloramphenicol. The cicatricial cases naturally required plastic procedures.

Leprosy with ocular involvement was also seen. Corneal involvement included superficial punctate keratitis, leproma (leprotic nodules) of the cornea, interstitial keratitis, loss of sensation due to fifth nerve impairment, and exposure of the cornea secondary to seventh nerve involvement (figs. 5-A, 5-B, and 5-C). Loss of eyelashes and typical cutaneous changes in the eyelids and adjoining skin were seen. Nodules were also seen in the sclera. Uveitis due to leprosy was also prevalent. Many of the patients suffering from leprosy were not isolated and we performed cataract extractions on some; the results were surprisingly good. Naturally, surgery was not performed in the presence of active uveitis.

Because vaccination is not universal in India and Pakistan, smallpox still exists. Corneal involvement, resulting in dense scars or perforation, frequently resulted so that this preventable disease is still an important cause of blindness in that part of the world (fig. 6).

Eye infections ranging from mild conjunctivitis to severe endophthalmitis and panophthalmitis seemed to be more common than in the United States. Glaucoma may have been more frequent due to the increase of untreated secondary types. Also the practice of couching of cataracts by some quacks resulted in patients being blind and suffering from glaucoma. It is noteworthy that no cases of retrolental fibroplasia were seen. This is one disease present in the United States but not present in India.

In summary, I have described the practice of ophthalmology as I found it in small villages of India and Pakistan. It may vary in the larger cities. However, since 85 percent of the population is concentrated in rural areas, I believe this report gives a more realistic picture of our specialty in that part of the world.

269 South 19th Street (3).

PREVENTION BY THYROXINE OF THE OCULAR CHANGES*

NORMALLY PRODUCED BY ββ′-IMINODIPROPIONITRILE (IDPN)

HANS SELYE, M.D.
Montreal, Canada

Earlier observations have shown that ββ′-iminodipropionitrile (IDPN), given subcutaneously, produces rather selective lesions in the eyes in several species of experimental animals (monkey, guinea pig, rat, rabbit, cat, dog, hamster). The character of these lesions varies according to the species. Thus, in the monkey, retinal changes are exceptional while retro-orbital and palpebral edema are always pronounced. In the rat, however, the most common lesions are: clouding of the cornea (often progressing toward severe keratitis), edema of the retina with degenerative changes in the inner and outer nuclear layers, and retinal detachment

* From the Institut de Médecine et de Chirurgie expérimentales, Université de Montréal.

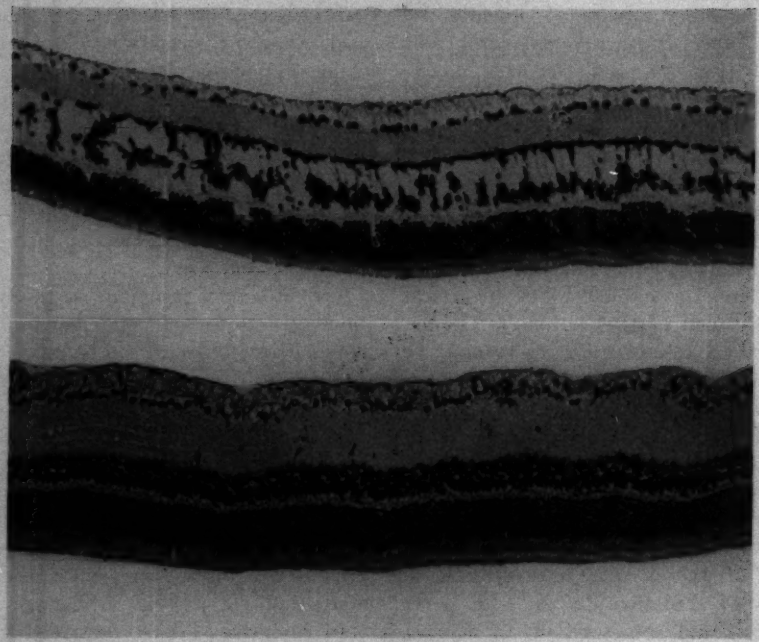


Fig. 1 (Selye). Retinal edema and the degenerative changes, which affect particularly the inner nuclear layer of the IDPN-treated animal (top). These changes are completely absent in the retina of a rat simultaneously treated with IDPN and thyroxine (hematoxylin-eosin, ×100).

(sometimes accompanied by hemorrhages into the aqueous humor).[1]

It is the object of this communication to demonstrate that treatment with thyroxine can completely prevent the ocular lesions that are normally produced by IDPN in the rat.

Materials and Techniques

Twenty female Sprague-Dawley rats, weighing 97 to 111 gm. (average 102 gm.), were subdivided into two equal lots: Group I received only IDPN; Group II, IDPN and thyroxine.

IDPN (ββ′-iminodipropionitrile, also known as aminodipropionitrile, or bis-[β-cyanoethyl]-amine) was administered subcutaneously, at the daily dose of 30 mg. in 0.2 ml. of water during 10 days. Then 100 mg. in 0.2 ml. of water were given during the next two days. On the 12th day of treatment, IDPN administration was discontinued, since all the animals of the group that received no thyroxine showed pronounced neurologic disturbances (excitation, choreiform movements, and "circling behavior") which are also characteristic consequences of intoxication with IDPN.

Thyroxine was administered in the form of its sodium salt at the daily dose level of 250γ in 0.2 ml. of water. Subcutaneous injections were begun four days prior to the IDPN administration and were continued until the fifth day after cessation of treatment with the nitrile. Pretreatment with the hormone was thought to be advisable, since many of the actions of thyroxine develop slowly and, therefore, marked thyroid hormone effects could not be expected for several days.

Results

During the five days following the interruption of the IDPN treatment, the usual

neurologic manifestations of IDPN-intoxication became extremely evident in the animals of Group I, and all 10 of these rats (treated with IDPN alone) eventually succumbed. On the other hand, in Group II (treated with IDPN plus thyroxine), the neurologic manifestations of IDPN intoxication were mild, and none of the animals died.

The eyes of all 20 rats were embedded in paraffin and examined on sagittal sections stained with hematoxylin-eosin. No evident histologic change was observed in any of the rats treated with thyroxine in addition to IDPN, while all the animals given IDPN alone showed keratitis, degenerative changes in the retina, and retinal detachment (fig. 1). In addition, four animals in this latter group exhibited marked hemorrhages into the aqueous humor.

These findings indicate that thyroxine is extremely effective in counteracting the toxic effects that IDPN normally exerts upon ocular structures. The mechanism of this protective effect remains to be elucidated. It is well known that changes in the thyroid hormone content of the blood can exert rather selective effects upon the eyes as exemplified by the exophthalmos of Graves' disease. Yet, it is unlikely that in our experiments thyroxine antagonized the effect of IDPN through some such selective action upon ocular structures, because the mortality as well as the systemic neurologic manifestations of IDPN were also inhibited by the hormone. It is more probable that thyroxine interferes with the effect of IDPN upon its chemical substrate in all tissues or that the thyroid hormone increases the rate at which the nitrile is detoxified within the body. In any event, these observations demonstrate, in the case of yet another experimental disease, the validity of the thesis[2] according to which hormones can play a decisive role in the genesis of nonendocrine diseases, that is, of maladies not primarily caused by an excessive or deficient function of an endocrine gland.

Summary

Experiments on albino rats indicate that keratitis, with degenerative changes and detachment of the retina, can be produced by ββ′-iminodipropionitrile (IDPN) with great regularity. All these changes are completely prevented by simultaneous treatment with thyroxine.

Case Postale 6128.

Acknowledgments

These investigations were performed with the aid of grants from the Warner-Chilcott Laboratories and from the Medical Research Board, Office of the Surgeon General, Department of the U. S. Army, Contract No. DA-49-007-MD-186. I am greatly indebted to the Abbott Laboratories for supplying generous quantities of ββ′-iminodipropionitrile.

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Ophthalmic Miniature

Spectacles. I don't use them. All I ask is a large, fair type, a strong daylight or gaslight, and one yard of focal distance, and my eyes are as good as ever.

Oliver Wendell Holmes,
The Autocrat at the Breakfast Table, 1858.

PERIVASCULAR GLIA OF THE BLOOD VESSELS OF THE HUMAN RETINA*

J. Reimer Wolter, M.D.
Ann Arbor, Michigan

The blood vessels of the human retina are surrounded by a very complicated system of cells and fibers. Around the arteries and veins a dense adventitia of connective tissue and processes of neuroglia (astroglia) form a coarse layer. Much more delicate structures are found around the precapillaries, capillaries, and postcapillary veins. The wall of these small vessels is formed by a single layer of flat endothelial cells and also contains different types of pericytes.[1] The pericytes are considered their contractible elements. Perivascular networks of delicate nerve fibers lie on the outside of the wall in close relationship to the endothelium and the pericytes. There are also endings of somewhat coarse nerves which come from the nerve fiber layer and represent the centrifugal nerves of the optic nerve and the retina.[2]

The astroglia of the retina is known to form a complete sheathing of the small blood vessels with their "sucker footlike" processes.[3,4] There is an actual space between the wall of these blood vessels and the glial sheath which is known as the Robin-Virchow space. The glial sheathing results functionally in a total separation between the mesodermal tissues of the blood vessels and the neurons of the retina. A destruction of this barrier results in degeneration of the surrounding retinal neurons and formation of a glial scar.

Between the sheath of astroglia and the structures of the small blood vessels themselves there is in the human retina a special cellular element. These cells were briefly mentioned in recent demonstrations[3,4] and then called "perivascular glia." However, very little is known about their nature and function. It is the purpose of this paper to give further histologic details about these peculiar cells and their bizarre changes under some pathologic conditions.

Material and method of histologic examination

Retinas of normal and pathologic human eyes were used for this study. The eyes were fixed in bromformalin (Cajal solution) immediately after enucleation or were injected with the same fluid shortly after death. Parts of the retinas were isolated and flat sections were made on the freezing microtome. These sections were stained with a silver carbonate technique of del Rio Hortega, the astroglia method as described by Scharenberg and Zeman.[5] Even with this technique it is not possible to stain the perivascular glia routinely. A certain amount of luck is necessary to impregnate these cells but not to stain the surrounding astroglia.

All illustrations presented in this paper are unretouched photomicrographs.

Histologic description

Figure 1 shows the high-power view of a branching precapillary in the degenerated retina of a 70-year-old man with secondary glaucoma following lens luxation. A perivascular glia cell of the kind here described can be seen directly upon the wall of this small blood vessel. Its body is star-shaped. It has a small round nucleus and long processes which surround the endothelial wall. This cell—although seen in a degenerated retina—is a good example of a normal element of this type.

Another normal perivascular glia cell is shown on a capillary of the normal retina of a 62-year-old-Negress who died of cancer (fig. 2). This cell has even more powerful processes which coil around the wall of the

* From the Laboratory of Neuropathology and Neuro-ophthalmology and from the Department of Ophthalmology of the University of Michigan Hospital. Supported by Grant No. B-475-C3 of the United States Department of Public Health, Education, and Welfare.



Fig. 1 (Wolter). Perivascular glia cell (arrow) with long processes on the wall of a precapillary of the retina of a 70-year-old man with secondary glaucoma following lens luxation. The connective tissue of the adventitia of an artery is visible in the left lower corner of the picture. (Hortega method, photomicrograph, ×1,000.)



Fig. 2 (Wolter). Perivascular glia cell (arrow) surrounding a capillary of the retina of the normal eye of a 62-year-old woman. The retinal astroglia is only partly stained. (Hortega method, photomicrograph, ×1,000.)

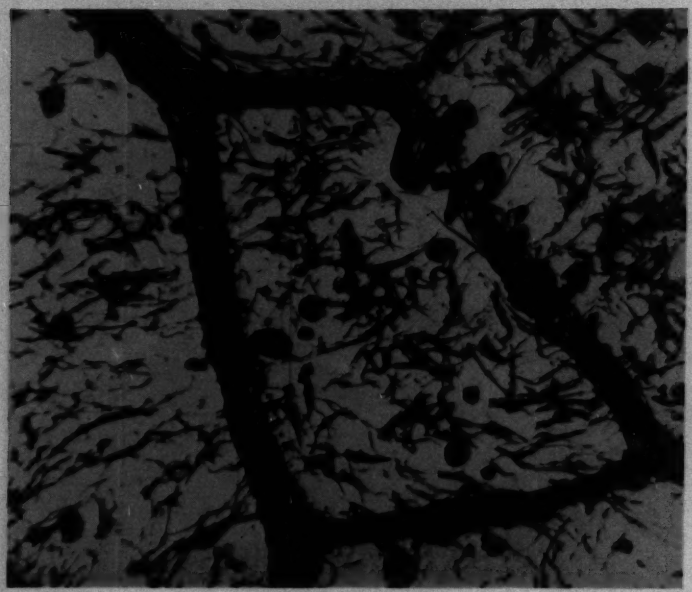


Fig. 3 (Wolter). Two hypertrophic perivascular glia cells (arrows) around a capillary of the atrophic retina of a 55-year-old patient with absolute, chronic, simple glaucoma. (Hortega method, photomicrograph, ×350.)

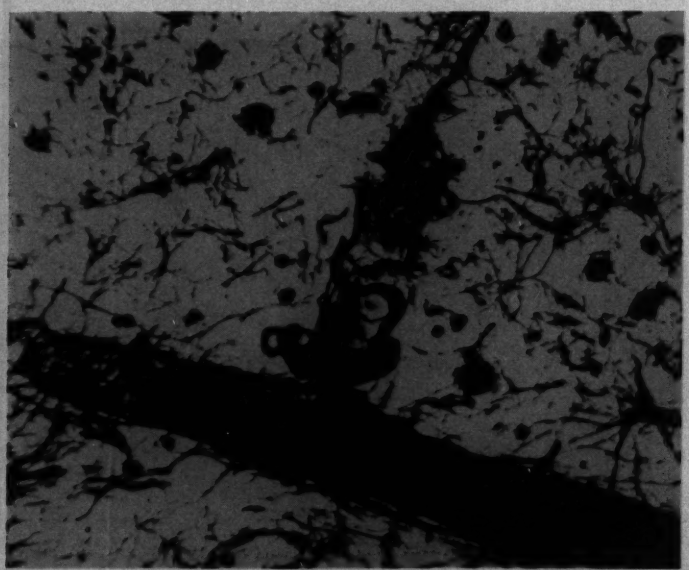


Fig. 4 (Wolter). A hypertrophic perivascular glia cell around a precapillary of the retina of a 55-year-old patient with secondary glaucoma following old iritis. (Hortega method, photomicrograph, ×500.)

blood vessel. It has also a small round nucleus. The surrounding astroglia is only slightly stained; this makes it possible to see the perivascular element very well.

Figure 3 is taken from a flat section of the atrophic retina of a 55-year-old patient with absolute, chronic, simple glaucoma. The eye was enucleated because of blindness and pain from degenerative keratitis. The photomicrograph shows an area of the capillary network of the inner plexiform layer. Two hypertrophic perivascular cells are visible on one of the capillaries (arrow). These cells represent coarse "snakelike" formations which coil around the vessel wall. The small round nuclei can still be recognized. These two perivascular cells show some hypertrophy as an obvious histologic change.

A similar but somewhat more severe situation with hypertrophy of such a perivascular cell is shown in Figure 4. The cell shows coarse, snakelike processes and coils around a small precapillary which branches off an artery. The picture is taken from the flat section of the retina of an eye with secondary glaucoma following old iritis.

Figures 5 and 6 represent not only hypertrophy but also advanced proliferation of such perivascular glia. Many long and coarse processes cover the wall of the small blood vessels and also invade the surrounding retinal tissue. The vessels probably represent precapillaries but are severely changed by extensive hyalinization. Figure 5 illustrates a section of an eye with old absolute glaucoma. Figure 6 represents a section from the degenerated retina of a blind eye with old retinal detachment.

A very extensive proliferation of the perivascular glia is observed in the retina of a 75-year-old man with advanced retinitis pigmentosa. The structures of all blood vessels in this case show different stages of degeneration and atrophy. But this atrophy is cor-

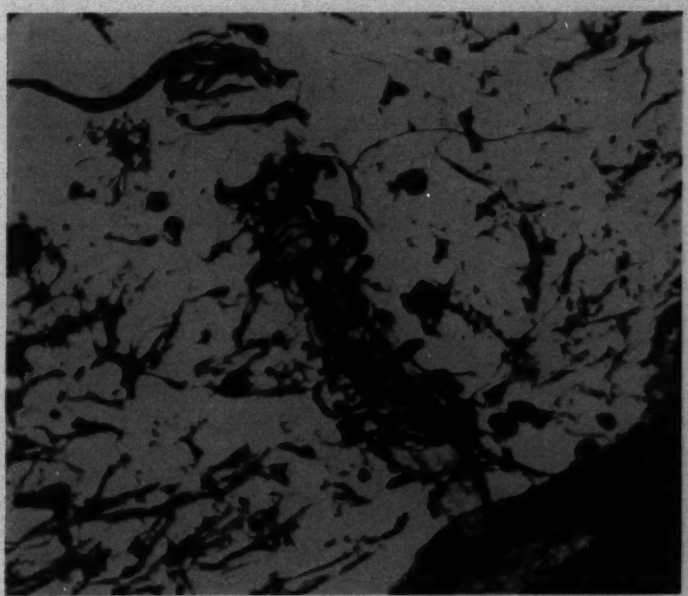


Fig. 5 (Wolter). A hypertrophic perivascular glia cell of the retina of a 72-year-old man with absolute glaucoma coils around a postcapillary vein and sends one process into the surrounding retinal tissues. Structures of the wall of a larger vein are visible in the lower right corner of the picture. (Hortega method, photomicrograph, ×500.)



Fig. 6 (Wolter). Hypertrophic and proliferating perivascular glia cells around two precapillaries of the retina of an eye of a 37-year-old man with retinal detachment. A round hyaline body (cytoid body) is visible in the right upper corner, and a part of the wall of the artery is shown in the left lower corner of the picture. (Hortega method, photomicrograph, ×500.)

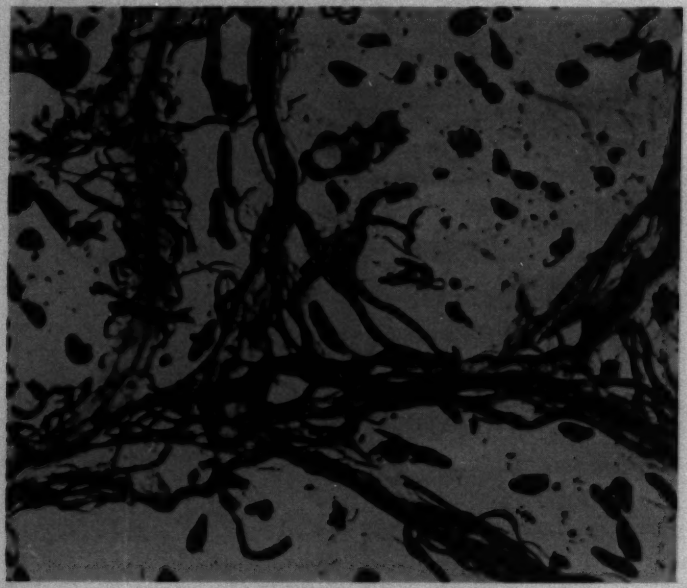


Fig. 7 (Wolter). An area with degenerated capillaries of the retina of a 75-year-old man with advanced retinitis pigmentosa. Proliferating perivascular glia surround the hyalinized structures of these blood vessels. (Hortega method, photomicrograph, ×400.)

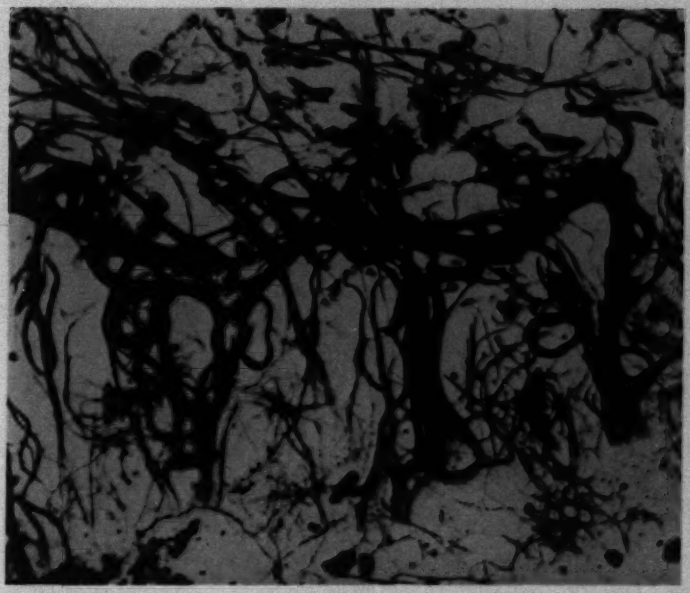


Fig. 8 (Wolter). Another area of even more degenerated capillaries of the retina of the 75-year-old man with retinitis pigmentosa. Only few remnants of the vessel wall can be seen. Proliferating perivascular glia form a dense structure around the remnants of the capillaries and send their processes into the retinal tissue. (Hortega method, photomicrograph, ×400.)

related with an extensive proliferation of the perivascular cells. The fewer structures of the vessel wall survived, the more hypertrophy of the perivascular glia can be seen around them. Figures 7 and 8 represent stages of vascular degeneration where there still are some hyalinized remnants of the vessels visible. The perivascular glia cells show an extensive proliferation and send their processes not only around the vessels but also into the surrounding retinal tissue. Figure 9 demonstrates that a dense interconnected network of hypertrophic perivascular cells remains in the retina after complete destruction of an area of the capillary network. The blood vessels are no longer visible, but the proliferated perivascular cells indicate where the capillaries were.

Discussion

This study confirms the existence of special perivascular cells which are closely attached to the wall of the small blood vessels of the human retina. These cells are distinctly different from the endothelial cells and pericytes of the vessel wall and from fibroblasts. They can also be clearly distinguished from the retinal astrocytes which form the glial sheathing around the small vessels.

The cells here described normally cover most of the wall of all small blood vessels with a loose network of their long processes. But they do not form a continuous layer. Under some pathologic conditions—especially primary or secondary degeneration of the retina—these perivascular cells show extensive hypertrophy and proliferation. They often survive the destruction of all vascular structures. Normally all processes of these cells are closely attached to the vessel wall. Under pathologic conditions, however, their hypertrophic processes extend into the scarified astroglia of the surrounding retina.

It is our impression that the perivascular cells here observed represent a special kind



Fig. 9 (Wolter). An area of the retina of the same old man with advanced retinitis pigmentosa which exhibits complete destruction of the capillary network. The proliferating perivascular glia remain in the retina and form an interconnected network which still indicates where the capillaries were. (Hortega method, photomicrograph, ×400.)

of retinal glia. This perivascular glia is found only on the walls of small blood vessels, and its function must therefore have to do with the retinal blood supply.

Liss[6] recently demonstrated exactly the same perivascular cells in the human olfactory bulb and also considered them to be glia. This indicates that the occurrence of such perivascular cells is not limited to the retina. Buno[7] interpreted similar perivascular elements in the bovine hypophysis as cells of endothelial nature (endothelios vasculares). But this latter concept is not satisfactory, since these cells always lie on the vessel wall—clearly outside of the endothelial layer. Furthermore, they have in contrast to endothelial cells small glialike nuclei and long processes. It must be mentioned, however, that these perivascular cells sometimes look similar to oligodendroglia as seen in other parts of the central nervous system. Further studies will be necessary for an understanding of the function and a final classification of the perivascular cells.

Summary

A special type of glia surrounds the small blood vessels of the human retina with long processes. These cells are called the perivascular glia. Hypertrophy and bizarre proliferation of the perivascular glia can be observed under primary and secondary degenerative conditions. Virtually nothing is known yet about the significance and function of these cells.

University Hospital.

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SCLERAL BUCKLING OPERATION WITH INSERTION OF CHROMIC CATGUT*

ANGELOS DELLAPORTA, M.D.
San Francisco, California

Previous experimental work[1] showed that a scleral buckling operation with the inclusion of a polyethylene tube 30 mm. long and 1.0 mm. in the external diameter produced a cylindrically shaped protrusion of the sclerochoroidal wall into the interior of the treated eye.

Immediately after surgery the protrusion was 1.0-mm. high and 1.0-mm. wide plus the thickness of the sclera and choroid enveloping the tube. The external opening of the scleral buckling was firmly closed eight to 10 days after surgery by young connective tissue so that by that time the polyethylene tube was well buried in the scleral tissue.

The size of the protrusion seen immediately after the operation remained practically unchanged for six to eight weeks; thereafter, it was found to be about one-third of the original size, so that the final effect was a slight unevenness of the inner surface of the eye along the operated area. This decrease of the original protrusion is due to the tendency of the sclera to reassume its normal shape and size and is observed also in the related scleral resection operation.

From these findings it was concluded that a curative effect of a scleral buckling operation for retinal detachment would most probably be due to the temporary protrusion of the sclerochoroidal wall. Since the inclusion of the polyethylene tube has only a temporary effect, while remaining there indefinitely sometimes causing undue irritation or even being extruded, the next logical step was to look for a substitute material which would produce the desired temporary protrusion for six to eight weeks but would then be absorbed, avoiding the undesirable foreign body reaction. Surgical absorbable catgut seemed to be the most promising material.

In order to get the necessary information experiments were carried out in 38 normal dog eyes using plain catgut or chromic catgut for inclusion into the scleral buckling and, as suturing material, chromic catgut or nonabsorbable silk. The surgically treated eyes were enucleated at times varying from 24 hours up to five months after the operation and the specimens were examined histologically. A detailed report of these experiments will be given in another paper.

Based on the experience obtained from these experiments, the following operation was developed in which chromic catgut is used instead of polyethylene. It consists of the following steps:

* From the Second Eye Department of the University of Vienna. This study was supported by the Buffalo Eye Bank and Research Society, Inc., Buffalo, New York. Read before the Ophthalmological Society of Vienna, March 11, 1957.

* Manufactured by Badische Anilin und Soda Fabrik A. G. (BASF), Ludwigshafen/Rhein, Germany.

1. When general anesthesia is administered, it is advisable to inject 1.0 to 2.0 cc. of one-percent procaine hydrochloride combined with adrenalin in the retrobulbar space. This causes a slight protrusion of the globe and prevents excessive bleeding from the conjunctival and episcleral vessels.

After external canthotomy the sclera is freed from the conjunctiva, the episcleral tissue, and the muscles, as in penetrating or lamellar scleral resection.

2. The equatorial strip of sclera, 3.0-mm. wide, which is to be infolded is outlined with the lancet caliper (fig. 1-A). The distance of the scleral strip from the limbus toward the equator and its length depend on the choice of the surgeon and the requirements of the case operated. The outlines of the strip are carefully deepened with a small Bard-Parker knife and simultaneously the scleral tissue of the strip is pushed with the point of the knife toward the middle of the

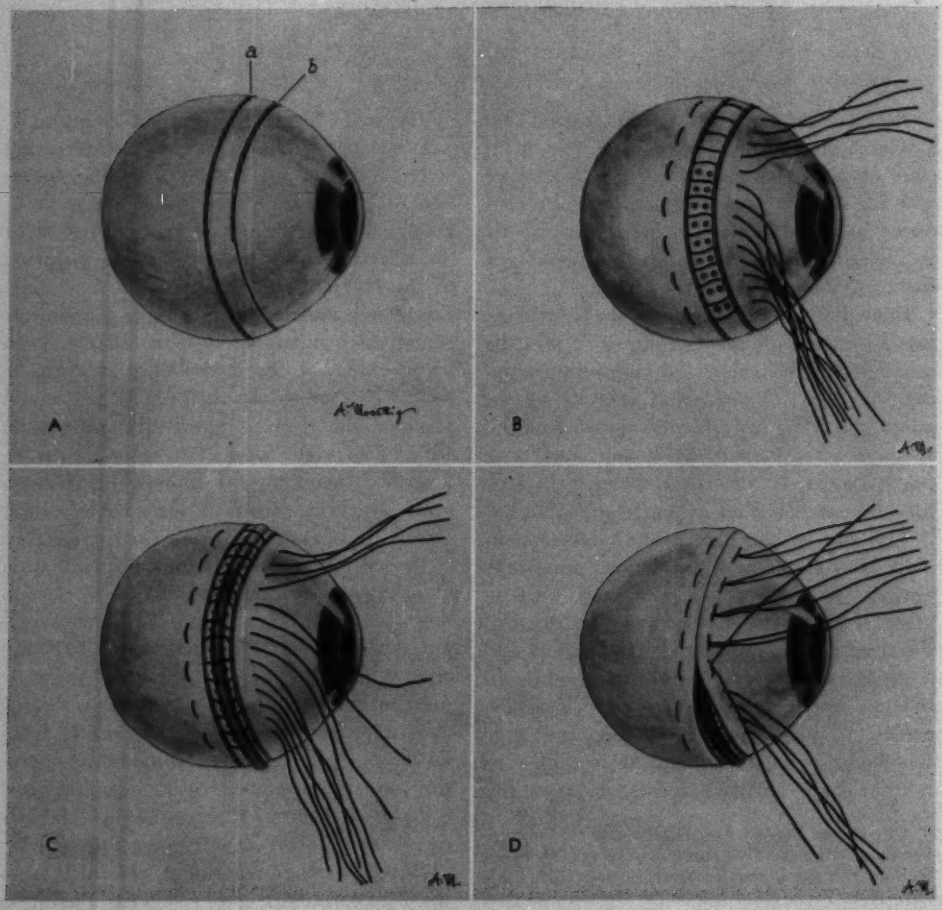


Fig. 1 (Dellaporta). Four steps of the scleral buckling operation with insertion of chromic catgut. (A) Outline (b) of the scleral strip is halfway deepened and widened. (B) Insertion of sutures and application of nonperforating diathermic coagulations on the scleral strip. (C) The chromic catgut thread is slipped under the sutures. (D) The sutures are tightened, the catgut is buried within the scleral fold; the scleral wound lips are in good apposition.

strip. This facilitates the deepening and widening of the outlines up to the innermost scleral layers which must not be perforated. The outlined scleral grooves represent anatomically real scleral wound lips (fig. 1-A).

3. Silk mattress sutures (3-0) are inserted as shown in Figure 1-B with 2.0- to 2.5-mm. wide intrascleral bites to obtain a good holding in the scleral tissue. The distance between the single threads of the inserted sutures should be about 1.5 to 2.0 mm.

Better than silk proved to be a synthetic suturing material called Supramid* (0.2-mm. thick). It is a braided thread and can be sterilized either by dry heat or by boiling in water for 30 minutes. The Supramid thread has a higher tensile strength, is flexible, and slides much more smoothly in the scleral tissue than silk, obvious advantages for the secure closing of the wound. Also it has a better hold on the tissue, since, being chemically inert, it produces less tissue reaction. Small sharp Grieshaber needles (No. 80/5a or 80/5b) facilitate greatly the placement of the sutures.

4. Diathermic nonperforating punctures are applied to the scleral strip in two rows with a 0.75- to 1.5-mm. long needle with insulated bases (fig. 1-B). The length of the needle depends on the thickness of the sclera, and the diathermic current should be of the same strength and should be applied for the same time as for producing ordinary, ophthalmoscopically visible transscleral coagulations. Diathermic current must not be applied in either of the scleral grooves in order to avoid damage of the scleral wound lips.

5. A piece of chromacized surgical catgut* equal to the length of the scleral strip is slipped under the sutures as shown in Figure 1-C. The diameter of the catgut must not be bigger than 1.0 mm. because the width of the scleral strip needed to envelop it is about 3.0 mm. The size relation between the inserted catgut thread to the width of the scleral strip should be 1:3 ($2 \pi r$).

The intraocular pressure is then lowered by drainage of subretinal fluid through several perforating diathermic coagulations applied over the region where the detached retina shows its highest elevation. If necessary, a small paracentesis is performed.

The sutures are then immediately tightened and knotted in sequence. During this procedure, the sclera infolds and the catgut thread are gradually buried within the fold as shown in Figure 1-D. The surplus catgut is cut off and the ends buried within the fold with two additional single sutures.

At the end of the operation the anterior and posterior scleral wound lips are in good apposition and should cover entirely the inserted catgut thread. Any small dehiscences should be closed by additional single sutures.

6. Finally the muscles and the wounds of the conjunctiva and the canthotomy are repaired.

Comment

For the success of the operation the following points are of importance:

1. To deepen and widen the outlines of the scleral strip in order to create real wound lips with good and quick healing tendencies.

2. To form the scleral strip wide enough to envelop entirely the inserted catgut, so that the scleral wound lips come into good apposition.

3. Not to apply diathermic current at the scleral wound lips so that the healing capacities of the scleral tissue remain unaffected.

If these steps are carefully observed the following is achieved:

1. The scleral wound closes by formation of connective tissue within four to six days after the operation.

2. The catgut thread is protected from the disintegrating action of the numerous inflammatory cells deriving from the inflamed episcleral and conjunctival tissue.

3. The catgut thread is subjected only to the disintegrating action of the inflammatory

*According to the U.S.P. specifications No. 5 nonboilable surgical gut has a diameter of 0.864 to 0.978 mm., No. 6 boilable surgical gut a diameter of 0.813 to 0.914 mm.

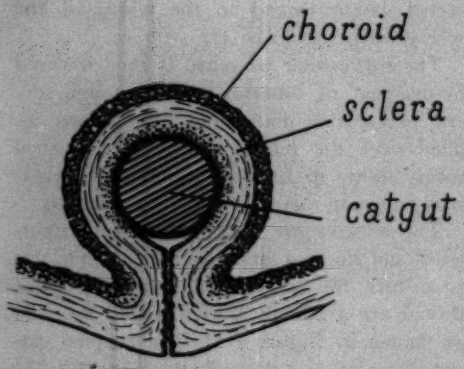


Fig. 2 (Dellaporta). The relation of the membranes of the eye around the catgut. The scleral wound lips are in good apposition.

cells deriving from the enveloping scleral strip. Their number is small since the scleral tissue in itself has very few vessels and in addition the enveloping scleral strip is damaged by the diathermic applications.

4. Therefore, the protrusion into the interior of the eye will be of long duration.

From our experimental data it is evident that if after tightening the sutures the wound lips remain 1.5 to 2.0 mm. apart in the entire length of the scleral buckling the protrusion of the sclerochoroidal wall will not last longer than three to four weeks.

No lamellar resection is done on the scleral strip so that the full thickness of the sclera enveloping the catgut thread contributes as much as possible to the size of the protrusion. An interesting new method of scleral buckling operation using the tissue of the scleral strip for the formation of the protrusion was reported by Chamlin and Rubner,[2] while my experimental studies were in progress. The method and the one presented here, compared to the scleral buckling operation with the use of a polyethylene tube, have the great advantage that finally no foreign material remains in the sclera. The only drawback of the method of Chamlin and Rubner is that, if the sclera of the operated eye is thin, the protrusion cannot be of the desirable size. This is avoided in the presented method by inserting catgut.

It is beyond the scope of this paper to discuss the indications for the scleral buckling operation or its merits in the treatment of retinal detachment. The only purpose of this presentation is to draw attention to the fact that, by replacing the polyethylene tube with a chromicized catgut thread in the scleral buckling operation and by observing specifically certain steps in the course of the operation, one achieves the same mechanical effects without the possible complications arising from tissue intolerance against the polyethylene.

Summary

Based on experiments on animal eyes a scleral buckling operation was developed and is described, in which the protrusion is caused by the insertion of a chromicized catgut thread.

2018 Webster Street (15).

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EXOTROPIC RETRACTION SYNDROME

With congenital homolateral ptosis

Burgos T. Sayoc, Lieut. Col. (MC) A.F.P.
Manila, Philippine Islands

Since textbook descriptions of the retraction syndrome seem to associate the condition with convergence strabismus,[1, 3, 4] whereas in my experience with the following two cases it is associated with divergence strabismus and is accompanied by congenital homolateral ptosis, this report seemed justified.

Case Reports

Case 1.

D. S., a single Filipino woman, aged 25 years, was examined on December 15, 1956 (fig. 1). She claimed the condition in her left eye has been present since birth. She had no complaint with her right eye.

Examination revealed: (1) Ptosis of the left lid; exoptropia of the left eye; (2) with the ptotic lid raised, the extreme exotropia and hypotropia were more clearly revealed; (3) on looking to the right, the direction of loss of movement, there was bulging of the left globe and the left eyeball could not quite be pulled to the vertical meridian; (4) looking to the left, there was retraction of the left globe and narrowing of the palpebral fissure; (5) oblique movement of the affected eye, down-and-out on either abduction or version toward the opposite side.

Vision, O.D., was normal for far and near, while, O.S., was counting fingers at two meters; Jaeger was negative.

It is generally recognized that if strabismus is present in cases of blepharoptosis, it should be corrected before surgical intervention for the latter condition. This assumes more significance with associated hypotropia, otherwise the danger of exposure of the cornea is greater. In spite of the presence of hypotropia in this case, I decided to deal with the horizontal muscles first with the assurance from the patient that she would submit to a second muscle operation three to four months later.

Operations. The first operation was done on December 20, 1956, under general anesthesia. The left lateral rectus was found inserted at a normal distance from the limbus. However, it appeared as



Figs. 1 and 2 (Sayoc). Case 1. (1) Before operation. (2) After operation.

a band of white glistening, inelastic fibrous tissue. It was recessed 10 mm., almost to the functional equator. The left medial rectus was found inserted at a normal distance, but appeared thin, well stretched, almost atrophied—a striking contrast to the broad, fleshy appearance of a normal medial rectus muscle. It was resected seven mm. Inasmuch as other muscle surgery could not be done for three to four months, the ptosis of the left upper eyelid was corrected on January 19, 1956. Due to the presence of hypotropia and the coexisting retractive syndrome, a simple corrective procedure for ptosis was used, that is, a modified Friedenwald-Guyton technique with simultaneous construction of the superior palpebral fold under local anesthesia.[3]

Results. Four weeks after the muscle operation, the motility of the eyes was tested in different directions of gaze. There was noticeable increase in the pull of the left eyeball to the right. What was striking was the persistence of the retraction syndrome in spite of the operation on the horizontal muscles. There was retraction of the left globe on abduction and slight bulging on adduction.

Six weeks after the muscle operation, the left eye could pull with the pupil just beyond the vertical meridian with slight bulging of the left globe. Looking straight ahead, the left pupil almost reached the vertical meridian (fig. 2).

After the ptosis operation, the retraction syndrome persisted but the left upper lid could be closed properly after the ptosis operation. Unfortunately, the patient said she felt so improved with the first stage that she could not be prevailed to submit to the second muscle operation.

Case 2

T. C., a single Filipino man, aged 45 years, was seen on September 20, 1956. According to him, his eye condition had been present since early childhood.

Examination revealed the left eye to be normal in all respects. The right eye was virtually shut due to almost complete ptosis of the right upper eyelid. On raising it, the right eye was in extreme exotropia with marked hypertropia on looking straight ahead. When looking to the left, the direction of less movement (adduction), there was noticeable bulging of the right globe with appreciable widening of the palpebral fissure. The right eyeball could hardly reach the vertical meridian. Looking to the right, there was appreciable retraction of the right globe with the subsequent narrowing of the palpebral fissure. An oblique movement of the affected eye, up-and-out, was observed either on abduction or on version toward the contralateral side.

Vision, O.D., was counting fingers at two feet; Jaeger, negative; while O.S. was normal for far and near.

Operations. On September 27, 1956, operation on the horizontal muscles, right eye, was done under general anesthesia. The right lateral rectus was found inserted normally. It appeared as a hard fibrous inelastic band, which was excised from its insertion and separated from check ligaments and adhesions to the sclera. The right medial rectus was found thin and almost atrophied. It was resected seven mm. On November 14, 1956, the ptosis on the right was corrected with a modified Friedenwald-Guyton technique.

Results. The operation on the horizontal muscle gave less improvement than in the first case. The right eye could hardly reach the vertical meridian when looking to the left (adduction). The ptosis operation gave only fair results.

Discussion

The retraction syndrome of the left eye, as described in textbooks, shows complete loss of abduction of the left eye, the same eye would retract upon adduction. Hence, when the patient looks to the left, the right eye would turn left, but the left eye would go only to the midline. On looking to the right, both eyes would go to the right but the left would retract slightly at the same time. This retraction of the left eye on looking to the right (adduction) would allow the homolateral upper lid to drop, thus producing some narrowing of the left palpebral fissure. There is often an oblique movement of the affected eye, either up and in or down and in when either adduction or a version toward the opposite side is attempted.[1,4] The retraction syndrome, be it associated with esotropia or exotropia, is characterized by similarity in its components which manifest themselves in an exactly opposite manner on adduction and abduction.

In these two reported cases, it should be noted that the first is an exotropic retraction syndrome with hypotropia. On the other hand, the second case has hypertropia so that, if either abduction or version toward the opposite side is attempted by the affected eye, the oblique movement of abduction is down and out while the version is up and out. In both cases, the retraction syndrome persisted in spite of recession of the lateral rectus to the equator in Case 1, and myomectomy in Case 2 and resection of the medial rectus of the affected eye in both cases.

According to Scobee,[1] the consensus is that surgery is of little benefit and even inadvisable in such cases. According to Wolff,

however, tenotomy of the medial rectus of one of his patients had no effect while, in two other cases, the results were better. He concluded in his paper that surgical interference may benefit some patients.[1] The benefits derived from the operation are: (1) restores partial adduction or abduction, as the case may be, and (2) does away with face turning toward the direction of the action of the paretic muscle.

Summary

These cases are presented (1) because there are conflicting opinions among ophthalmic surgeons on the benefits of surgical intervention in cases of the retraction syndrome (the results observed in these two cases may lend support to those who believe in the possible benefits of surgery); (2) the retraction syndrome, as described in textbooks, is presumably associated with esotropia, while the two cases reported are associated with exotropia with ptosis of the homolateral lid.

1980 Azcarraga Street.

Acknowledgment

I am indebted to Dr. Derrick Vail and Dr. Arnold A. Albright for their suggestions.

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RADICAL TARSECTOMY*

In the treatment of the cicatricial complications of trachoma

John A. Pratt-Johnson, F.R.C.S.
Johannesburg, South Africa

Introduction

Trachoma is said to be the commonest cause of blindness in the world today. In South Africa there are areas over which the incidence and severity of trachoma equal the worst in the world.

In Sekhukuniland, a native reserve in the North Eastern Transvaal, the incidence of trachoma is 100 percent of African Negro population over the age of five years.[1] Approximately seven percent of the 160,000 Africans living in Sekhukuniland have trichiasis complicating their trachoma.[1]

The magnitude of this problem is easily grasped, particularly when experience with these primitive people and their environment place long-term treatment and systematic follow-ups beyond the bounds of practicability. This paper attempts to demonstrate the important place that radical tarsectomy should occupy in the treatment of the cicatricial complications of trachoma in this type of environment.

In Sekhukuniland one is faced with the problem of primitive patients seeking treatment in the final stages of trachoma. The patients expect quick treatment and are loath to stay in hospital for longer than a few days. The radical operation described in this paper tries to remove surgically as much diseased tissue as possible and yet preserve good function and reasonable cosmetic appearance. The operation may be performed as an outpatient procedure and the method of suturing is particularly designed to simplify removal of sutures by untrained personnel.

Figure 2 adequately demonstrates the obvious shortcomings of a tarsectomy performed from the conjunctival approach and

* The material for this study was obtained at Jane Furse Mission Hospital in South Africa.

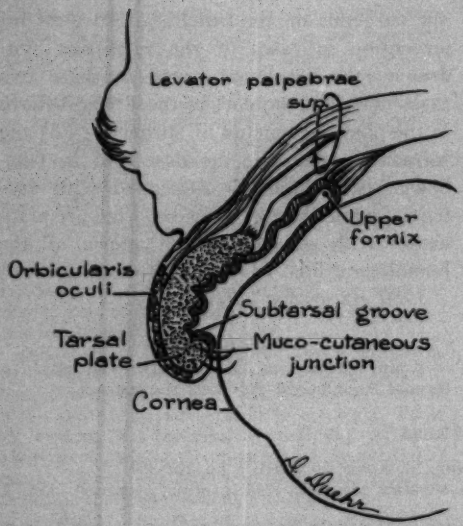


Fig. 1 (Pratt-Johnson). Vertical section of upper lid, showing a deformed thickened tarsal plate with irregular conjunctival surface, trichiasis caused by cicatricial entropion, and lashes emerging abnormally because their normal exit is blocked by fibrous tissue.

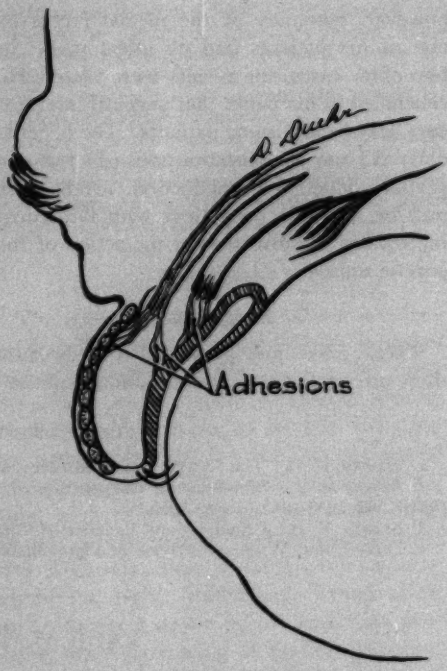


Fig. 2 (Pratt-Johnson). This shows the inadequacy of the classical tarsectomy by the conjunctival approach. Note passive entropion and no everting tendency.

according to the method described in most textbooks.[2]

It has been my experience that this operation produces no everting tendency and indeed many cases result in a passive entropion accentuated when the patient looks up. For this reason, the technique described in this paper was devised.

Principles of modified technique

In order to create an everting tendency to cure the entropion and trichiasis, the conjunctiva after excision of tarsal plate is transposed across the fulcrum of the lid margin and sutured to the lid anteriorly in the area of the orbicularis. This effectively everts the lid margin. An everting tendency on elevation is created by reattaching the levator palpebrae superioris to the skin as the skin incision is closed. Advantage is taken of the accentuated subtarsal groove in these cases by placing the distal tarsal incision at the anterior apex of the groove. This ensures that a rounded border contacts the cornea even in extreme eversion (fig. 3).

Operation

One traction suture is placed in the middle of the lid near the lid margin. The lid guard is inserted to protect the globe of the eye. An incision is made three mm. above the lid margin along its entire length. This incision reaches the tarsal plate hence dividing orbicularis as well as skin. The orbicularis is retracted upward and the whole tarsal plate is exposed (fig. 4). Using a No. 15 blade in a Bard-Parker knife, the conjunctiva is cut parallel to the upper border of the tarsal plate along its entire length. The tarsal plate and overlying conjunctiva are excised using a strong pair of straight scissors. It is most important that the incision around the distal margin lies at the deepest part of the accentuated subtarsal groove. The traction suture is

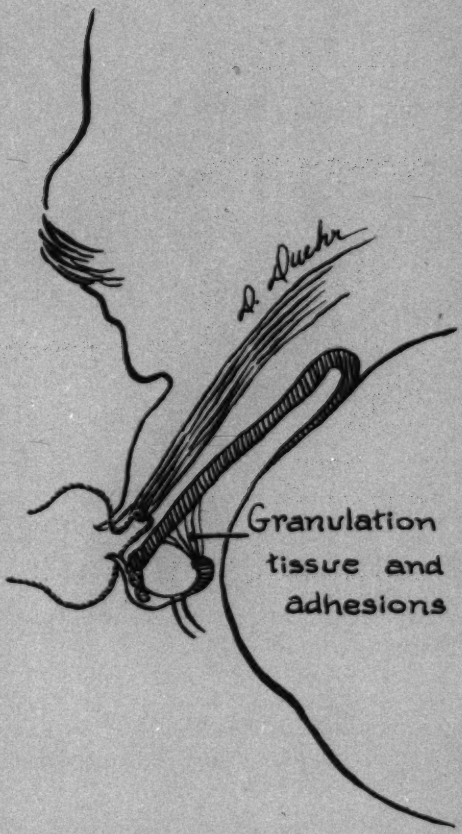


Fig. 3 (Pratt-Johnson). Demonstrates the advantages of the modified technique which ensures the marked eversion of the lid and everting tendency on elevation so essential in the management of this type of case.

cut and removed. The remaining conjunctiva, held taut with fixation forceps, is dissected free from its attachments at the fornix with scissors and further mobilized to the limbus. However, at the lateral extremity the conjunctiva is freed only sufficiently to facilitate suturing. This minimizes damage to lacrimal ductules and, if anything, enhances the effect of the operation by exerting an added everting tendency on the lateral margin of the lid where the entropion is most likely to recur.

The conjunctiva is now sutured to the lid margin just beneath the skin; No. 1 black



Fig. 4 (Pratt-Johnson). The skin has been incised and incision extended through the orbicularis muscle to the tarsal plate. The dotted line shows area of tarsal plate to be excised.

silk is used with a continuous key-pattern suture method (fig. 5). The ends are brought out at the extremities of the skin incision and are left four inches long. The distal margin of the levator palpebrae superioris muscle is included in the vertical component of a loose continuous key pattern suture with 3-0 white silk which closes the skin (fig. 6). These ends are also left long. A small acriflavine-gauze dressing is applied and is secured by bringing the suture ends over the dressing to the forehead where they are stuck down by selotape. Appropriate oculenta are inserted. The dressings are removed after 24 hours and the sutures on the fifth postoperative day by simple traction on one end of the suture.

Modifications

I. Elderly patients

When there is an excess of lax skin in the upper lid, an appropriate ellipse of skin should be excised.

II. Lower lid

In five percent of the entropion cases seen, the lower lids are affected. Wheeler's op-

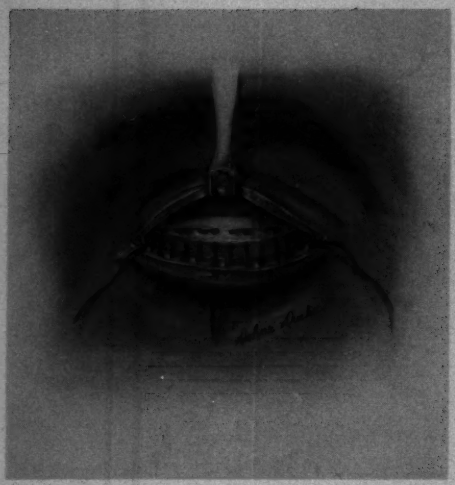


Fig. 5 (Pratt-Johnson). The tarsal plate has been excised and the conjunctiva is being sutured to the anterior part of the lid margin in region of orbicularis remnants by continuous key-pattern suture technique.



Fig. 6 (Pratt-Johnson). The final stage of the operation is shown. The levator palpebrae muscle is being incorporated in the continuous key-pattern suture which closes the skin.

eration was a failure in three out of four cases in which it was performed. Anterior excision of the lower tarsal plate combined with a skin and muscle operation produced perfect results in three cases and may be the operation of choice.

Results in this series

I have performed 170 operations for the cicatricial complications of trachoma. Over 100 of these have been the radical operation described in this paper. This operation has given the most satisfactory results but, unfortunately, the longest follow-ups have been only six weeks owing to the fact that the patients disappear into the vast expanse of bush.

Complications

Recurrence of trichiasis. Four cases recurred, three of these only at the lateral margin.

Dry eye. One case showed this condition.

Ischemic slough. One case in this series developed a slough of the middle one third of the lid. A too tightly pulled skin suture was thought to be the cause.

Infection was seen in one case which was mild.

Lid movements. All patients (except cases of lid margin slough) were able to open and close their eyelids satisfactorily.

Summary

Difficulties encountered in the treatment of trachoma in primitive people are mentioned.

It is argued that radical tarsectomy offers the best answer to the cicatricial complications.

Important modifications in technique are given. Results and complications in over 100 cases are discussed.

Stanford University Hospital,
San Francisco 15, California.

I would like to express my thanks to Dr. W. Downing, superintendent of the Jane Furse Mission Hospital in Sekhukuniland, for all facilities so generously provided and his whole staff for their kind co-operation.

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PITUITARY CYST

ALBERT A. STEINER, M.D.
San Francisco, California

The diagnosis of pituitary tumors depends essentially on three main findings: metabolic dysfunction, visual field defects, and radiographic distention of the sella turcica.

The purpose of this paper is to present a patient showing all three classical symptoms, whose treatment was unusual and whose postoperative courses were stormy. During the treatment of this patient, two new measures were introduced: (1) Visualization of the cyst with a radiopaque fluid (Pantopaque); (2) the use of radioactive cobalt, Co^{60}. Both substances were introduced into the cyst via the transseptal, transsphenoidal route.

CASE REPORT

Mrs. T. T. (G. E. H. #133557), a 51-year-old Japanese woman, was first examined at Greens' Eye Hospital on October 28, 1954. She stated that during the past month or so she had had a rather sudden decrease in vision in the right eye, and that she could not see well to the right or left with the right eye. Three weeks ago she had a sudden episode of sore throat, stuffy nose, fever to 103°F., and bilateral frontal headache. The fever subsided after several days of penicillin therapy but headache persisted. She had had mild dyspnea on exertion for five years but denied weakness, fatigue, dizziness, coldness, constipation, muscle pains, or paresthesias. She had lost about 100 pounds in the last two years.

Corrected visual acuity was: R.E., 20/60; L.E., 20/20—. Examination of the right eye showed the media clear, with slight pallor of the temporal side of the disc. Media and fundus of the left eye were normal.

Visual fields (Field 1). Right eye: Temporal hemianopsia with the nasal portion reduced to less than a 10-degree circle on the tangent screen, with a 20-mm. white test object at two meters. Left eye: Upper temporal field cut with a five-mm. white test object.

Skull X rays (Fig. 1). The floor of the sella is depressed. The posterior clinoids are thinned. There is some enlargement of the pituitary.

A diagnosis of pituitary tumor was made, probably a chromophobe adenoma, and she was referred to a neurosurgeon who, after careful examination, hospitalized her for pituitary surgery. However, because of the metabolic aspects of the case, an endocrinologist was called in consultation. After complete examination and many laboratory tests, he stated that the patient showed signs of adrenocortical and thyroid deficiencies.



Fig. 1 (Steiner). Skull X-ray film on October 28, 1954.

Operation for pituitary tumor is rarely justified unless visual symptoms are present. In preparation for surgery the patient received 100 to 200 mg. of cortisone intramuscularly daily for five days. At the end of this time she was more alert, had improved appetite, and the blood pressure in the erect position rose from 80/60 to 110/70 mm. Hg. Caution was exercised in the administration of preoperative analgesics and sedatives; the dose being no more than half of that usually given.

Surgery was performed on November 9, 1954, through a right transfrontal approach and revealed a cystic intracellar tumor pressing on both the right and left optic nerves, but particularly on the right. Aspiration yielded five cc. of a yellow viscid fluid containing a few flecks of whitish material. The cyst wall was very adherent to the under surfaces of both optic nerves and it was impossible to free them by dissection. Attempts to remove portions of the cyst wall with biting instruments resulted in dangerous traction upon the nerves. Therefore, the procedure was limited to destruction of the major portion of the cyst roof by electrocoagulation.

During the procedure the patient received intravenous glucose, saline, and hydrocortisone. Her condition at the end of the operation was good but a few hours later she complained bitterly of coldness and vomited several times. The next day her temperature was 40°C., with marked dehydration, despite liberal quantities of saline, glucose, intravenous hydrocortisone, and supplementary intramuscular desoxycortisone.

On the third postoperative day, she developed excessive thirst and the urinary volume, although never exceeding the fluid intake, seemed excessive considering the fever and clinical dehydration. Urinary specific gravity was 1.002. Fever and dehydration disappeared dramatically on a brief course of intramuscular pitressen tannate. She was dismissed from the hospital on a daily medication of cortisone (37.5 mg. in three divided doses), desiccated thyroid (gr. 0.1), and desoxycorticosterone linguets (2.0 mg).

During the next two months the dose of thyroid was gradually increased to 1.25 gr. daily, and her signs of myxedema disappeared.

Pathologic examination of the cyst fluid showed no tumor cells, but it was felt that the fluid probably originated within an adenoma.

Her return visit to Greens' Eye Hospital was November 29, 1954. Visual acuity was unchanged, but the visual fields were greatly improved (Field 2). The central fields showed a right temporal defect, but required a two-mm. white test object at two meters, and a slight upper temporal field defect in the field of the left eye with a two-mm. white test object.

One month later, December 29, 1954, visual acuity was the same, and the visual fields showed a slight increase in the temporal field defect of the right eye only. By January 3, 1955 (Field 3), the temporal field defect of the right eye was more marked on the tangent screen and perimeter.

The patient was again referred to the neurosurgeon who hospitalized her. However, because of her stormy course following intracranial surgery and because the pituitary lesion was known to be cystic, it was deemed advisable to attempt pituitary drainage via the transseptal, transsphenoidal approach. Therefore, an otorhinolaryngologist, experienced in this type of surgery, was consulted. In addition, an attempt was made to prevent further re-accumulation of fluid by irradiation of the cyst wall with an interstitial point source of radioactive cobalt, Co^{60}. This was performed January 13, 1955.

Two cc. of thick, opalescent, gelatinous fluid were removed and replaced with Pantopaque, enabling visualization of the cyst (fig. 2-A and B). A 200 mc. radioactive cobalt, Co^{60}, $1 \times 1 \times 1$ mm. encased in a 1.8 mm. diameter stainless steel shell, was inserted into the cyst at the end of a stainless steel wire, and each nasal chamber was packed with vaseline gauze to approximate the septal flaps against the wire.

Its initial position, determined by radiography, was found to be too far cephalad (fig. 3). It was retracted to a more satisfactory position (fig. 4).

Two hours later the cobalt, Co^{60}, was withdrawn after delivering a dose calculated to be 5,400 r at one cm. from the source center. The polyethylene tube was left in place, (fig. 5-A and B) to promote drainage and prevent subsequent sealing off of the surgical ostium.

Again her postoperative course was extremely stormy, consisting of moderate hypotension, vomiting, somnolence, dehydration, right lower lobe pneumonitis, and fever. On the second postoperative day thirst and polyuria ushered in the presence of permanent diabetes insipidus. Once again she responded dramatically to intramuscular pitressin tannate and reduced cortisone dosage. Her condition was critical for three days, following which she recovered gradually and was discharged on a daily dose of cortisone (25 mg. desoxycorticosterone, 1.0 mg.), thyroid (1.25 gr.), and posterior

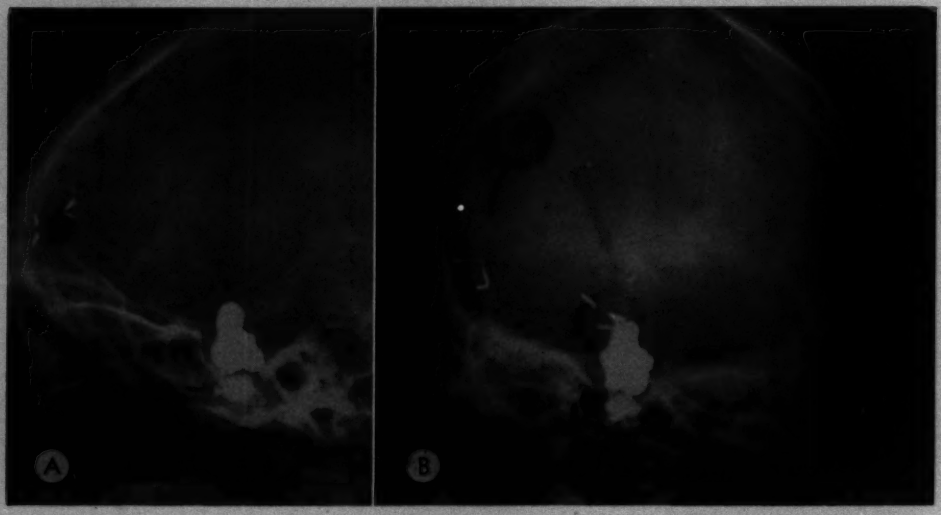
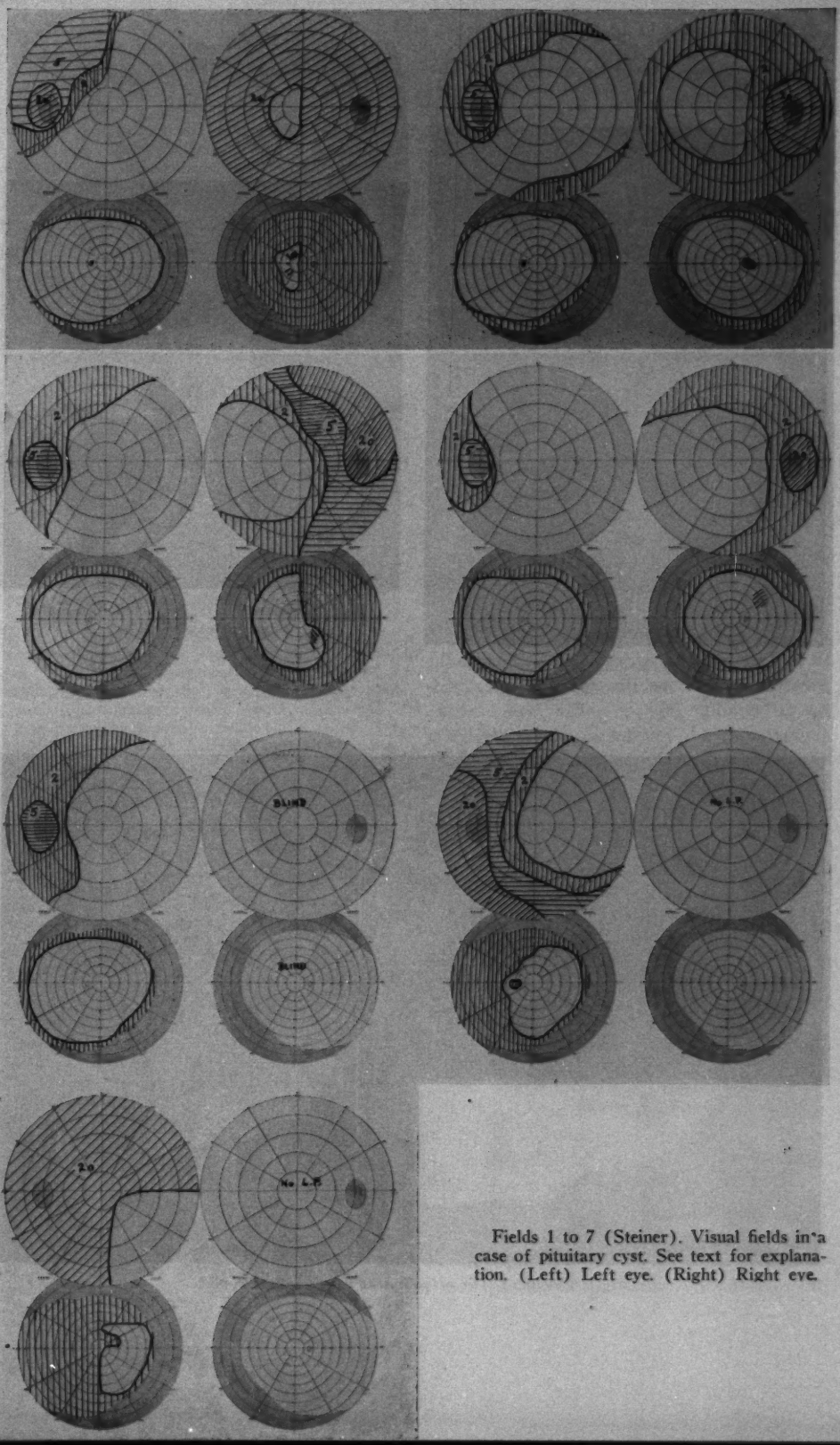


Fig. 2 (Steiner). Visualization of the cyst after Pantopaque injection.



Fields 1 to 7 (Steiner). Visual fields in a case of pituitary cyst. See text for explanation. (Left) Left eye. (Right) Right eye.



Fig. 3 (Steiner). Radiography showed the Co^{60} too far cephalad.



Fig. 4 (Steiner). Co^{60} retracted to a more satisfactory position.

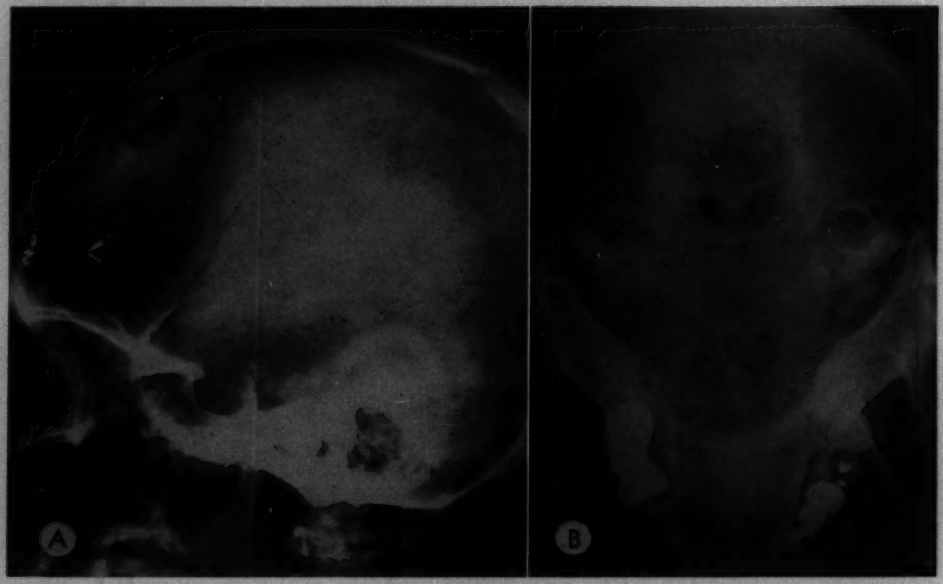


Fig. 5 (Steiner). The polyethylene tube was left in place.

pituitary powder by nasal insufflation as needed.

Her next visit to Greens' Eye Hospital was February 15, 1955. Vision in the right eye was 20/30— and in the left eye 20/20—. The discs were unchanged from the first visit. The visual fields (Field 4) again were much improved, requiring smaller-sized white test objects to elicit a temporal field defect.

At monthly intervals her vision, fields, and optic discs were examined. (The polyethylene drainage tube came out accidentally after several weeks.) They remained unchanged until November 22, 1955, when the patient reported that one week previously, the vision in the right eye was suddenly lost. (Field 5). Examination showed no light perception in the right eye. The pupil reacted consensually, but not directly to light. There was increasing pallor of the right optic disc. The visual field in the left eye was unchanged. A temporal field defect for a two-mm. white test object could be elicited on the tangent screen.

For the third time she was referred to the neurosurgeon and again hospitalized. The otolaryngologist was asked to drain the cyst as he had done before. On November 23, 1955, the transsphenoidal approach under local anesthesia was attempted. The cyst wall was no longer identifiable. Some clear fluid was aspirated. Radiography revealed the needlepoint to be in the third ventricle. The fluid was cerebrospinal. Despite this relatively minor surgical manipulation, the patient barely survived a stormy postoperative period lasting one week.

The patient was next examined at Greens' Eye Hospital on December 9, 1955. The right eye was blind with increasing pallor of the disc. The left eye was normal. The pallor of the right optic disc increased. The left eye remained normal and was examined at monthly intervals until November 19, 1956, at which time the patient noticed a decrease in vision on the left temporal side. The vision was 20/20 — 2. The field (Field 6) showed a temporal defect on the tangent screen and perimeter. The disc was normal.

The patient was referred to the neurosurgeon for the fourth time and was hospitalized. It was decided from the medical point of view that the only method of cure would be another transfrontal approach, since by the last transsphenoidal approach the cyst could not be visualized.

Because of her three previous operations, followed by extreme postoperative collapse, with danger of death, the family of the patient decided that no further surgery should be done. Therefore, she left the hospital without any surgical procedure having been performed.

On January 10, 1957, she was examined at Greens' Eye Hospital. The vision in the left eye had decreased markedly to finger counting at six inches. The remaining field (Field 7) was a small nasal island. The left disc now showed moderate optic atrophy. By April 23, 1957, vision in the left eye was reduced to light perception in the small nasal island.

Comment

The treatment of an expanding pituitary cyst requires the combined efforts of a group of specialists who must solve the following problems:

1. Relieve the pressure on the neighboring structures, especially the optic chiasm and the optic tracts.
2. Remove, cure, or control the lesion itself.
3. Treat the disturbance of associated and organ endocrine function.

Any given case may present signs and symptoms involving one of these categories and occasionally, as in the present case, all categories require consideration. There would appear to be no quarrel with the practice of recommending surgical decompression of the pituitary region when sight is threatened because radiation therapy may be too slow and medical therapy is not effective in shrinking pituitary tumors. However, there exists some difference of opinion as to whether one should employ the transfrontal or transsphenoidal approach. It is not the purpose of this paper to argue this, but rather to present a case in which both techniques were utilized and a third one developed.

The transfrontal approach for the first operation was best because it offered the opportunity of direct visualization of the tumor and adjacent structures, as well as the chance to obtain a biopsy and aspiration site. When symptoms of pressure returned, the transsphenoidal route was employed. However, aspiration alone was not enough. Irradiation of the lining of the cyst wall would be necessary in order to prevent the re-accumulation of fluid. Whereas, external irradiation by X rays (proton beams or linear accelerator) might be a statisfactory method of treating a solid pituitary tumor, a cystic tumor might better be treated by interstitial or intracavitary radiation.

The placement, particularly fixation of the

radon seeds, offered too many technical difficulties, with prospects of poor distribution of radiation and local necrosis. Interstitial irradiation from a point source of radium or Co^{60} within the cavity seemed logical. The choice of Co^{60} was determined by the availability of the material. The optimum dose and time exposure for treatment of cystic lesions must await analysis and treatment of other cases. A more fractionated course of radiation therapy might have been desirable. However, the 200 mc. source was the only one available at the time.

The violent postoperative reactions experienced by this patient are not usually seen in other cases, especially those in which there exists a disturbance of anterior lobe function only. In the present case there probably was functional impairment of the posterior lobe and hypothalamus.

The later failure in vision in the right eye may have been due to the effect of radiation on the blood supply to the right optic nerve, or it may have been due to a rapidly expanding cyst pressing on the right optic nerve. However, no expanding lesion was found at the third operation. Since no tumor tissue was obtained from the cyst wall, it cannot be decided whether the original lesion was a craniopharyngioma, cystic degeneration of a chromophobe adenoma, or some other type of lesion.

Summary

1. An interesting case of a pituitary cyst is presented which was diagnosed by the ophthalmologist and treated by the neurosurgeon, otolaryngologist, radiologist, and endocrinologist. Careful follow-up study was done for 28 months during which time the patient lost all her sight.

2. The pituitary cyst was operated on once via the transfrontal approach and twice via the transsphenoidal approach.

3. Two new measures were introduced: (a) Visualization of the cyst with a radiopaque fluid; (b) use of radioactive cobalt, Co^{60}, as a point source of interstitial radiation. Both substances were introduced into the cyst via the route of the nasal septum and the sphenoid sinus.

4. Careful endocrine evaluation prior to surgery was made. Adrenal-cortical insufficiency must be corrected prior to surgery. Caution must be exercised in pre-anesthetic medication in the myxedema patient. The possibility of cortical postoperative reactions may occur, particularly disturbances in thermal regulation, adrenal crises, and diabetic insipidus.

Greens' Eye Hospital,
Bush Street at Octavia.

I wish to thank Dr. Grant Levin, Dr. Shirley H. Baron, Dr. Leland R. Felton, and Dr. Joseph Kriss for their co-operation and assistance.

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OCULAR MANIFESTATIONS OF WEGENER'S GRANULOMATOSIS*

BRADLEY R. STRAATSMA, M.D.
New York

In recent years there has been increasing interest in a syndrome characterized by necrotizing lesions in the respiratory tract, diffuse arteritis, and focal glomerulonephritis. Undoubtedly, this interest has stemmed from the widespread tissue alterations of this fatal disease and the broad spectrum of pathologic alterations has diverted attention from the involvement of ocular structures. However, the ophthalmic aspects of this rare and obscure disease merit consideration because of their basic clinical interest, their position in the differential diagnosis of more common conditions, and their possible relationship to other poorly understood ocular disorders.

To obtain a proper perspective of these ocular features, a basic outline of this syndrome must be reviewed. The first description of this condition was presented in 1931,[1] but the formulation as a distinct entity awaited the careful study of three cases by Wegener in 1936[2] and 1939.[3] Therefore, it is reasonable to refer to this entity as Wegener's granulomatosis until additional fundamental knowledge permits a more descriptive title. Following Wegener's definition of this syndrome, numerous reports appeared in the literature including a comprehensive treatise by Godman, Fahey, and associates in 1954.[4,5] These authors summarized 22 cases from the literature and added seven new cases. Since then 15 additional cases (table 1) have been reported so that there is a total of 44 documented case studies available for review.

Several patients reported in the general[15–17] and ophthalmic literature[18] are probably examples of Wegener's granulomatosis but are atypical or reported in insufficient detail to be unequivocally accepted. Only confusion and misleading conclusions can result from failure to adhere to strict definition and adequate documentation in assembling cases. Consequently, the 44 patients accepted as a basis for this report are cases that have been verified anatomically by autopsy.

Reviewing these documented cases, it is evident that the natural history of the disease forms a striking clinical picture. There is no particular sex preponderance and these patients range in age from 12 to 70 years.

The first symptoms are generally referred to some part of the respiratory tract. Frequently the symptoms of nasal obstruction, discharge, and pain lead to the detection of rhinitis or sinusitis. In other cases a persistent productive cough, hemoptysis, and pleuritic pain focus attention on pulmonary pathologic processes. All combinations of pulmonary and upper respiratory involvement are encountered and variable constitutional symptoms are present. Regardless of therapeutic measures, these lesions tend to progress with perforation of the nasal septum and destruction of the bony confines of the paranasal sinuses. Breakdown of the in-

TABLE 1

DOCUMENTED CASES OF WEGENER'S GRANULOMATOSIS PUBLISHED SINCE THE REVIEW BY GODMAN AND FAHEY[4,5]

Case	Reference	Age (yr.)	Sex
1	6	31	M
2	7	middle	F
3	7	48	F
4	7	41	F
5	8	45	F
6	8, 9	12	M
7	10	41	F
8	11, 12	42	F
9	12	34	F
10	12	33	M
11	12	39	M
12	13	24	F
13	14	43	F
14	14	62	M
15	14	42	M

* From the Institute of Ophthalmology of the Presbyterian Hospital.

flammatory masses in the lungs may produce multiple pulmonary cavities.

The patient pursues a progressive or remittent downhill course accompanied by fever, leukocytosis, weight loss, and wasting. Diffuse dissemination of the disease is indicated by polyarthritis, dermatitis, carditis, and neuritis. Rather late in the course, albuminuria and retention of nonprotein nitrogen often appear. Indications of renal insufficiency may dominate the terminal phase and severe uremia generally precedes death.

The over-all course of the disease is usually two to five months in duration. Rarely, a remittent course lasting up to three years has been noted,[5] but all recognized cases have terminated fatally. Extensive studies have failed to implicate any micro-organism in the etiology and antibiotics seem only to control secondary infection. Therapy with systemic ACTH and adrenocortical steroids has been employed in most recent cases and has generally produced temporary improvement in symptoms and granulomatous lesions.

The diagnosis of Wegener's granulomatosis has been made during life with the aid of biopsy studies, but the diagnosis may be conclusively established only by post-mortem examination. Every case of Wegener's granulomatosis has three characteristic pathologic features—necrotizing granulomatous lesions in the respiratory tract, generalized focal arteritis, and necrotizing thrombotic glomerulitis.[4,5]

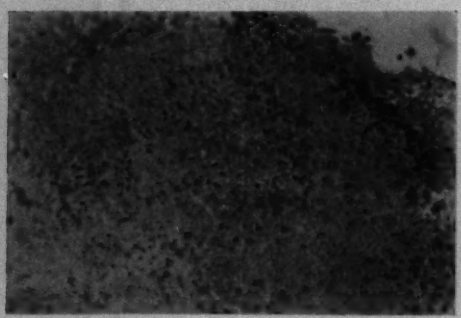


Fig. 1 (Straatsma). Maxillary sinus. Necrosis of the epithelium and underlying tissue. (A26618. Hematoxylin-eosin, ×60.)



Fig. 2 (Straatsma). Maxillary sinus. Viable tissue with intact segments of pseudostratified respiratory epithelium and an intense inflammatory response. (A26618. Hematoxylin-eosin, ×100.)

A composite view of these alterations indicates that the gross changes in the upper respiratory tract are related to widespread necrosis and inflammation of the respiratory mucosa. The membranes lining the nose and sinus spaces are shaggy, gray in color, and covered with a tenacious yellow purulent exudate. Extension of this aggressive necrotizing process frequently results in destruction of underlying bone and cartilage with collapse of the nasal arch and broad fistulas between the nose and sinus cavities. Further extension through the sinus walls may result in encroachment on the orbit, buccal cavity, or temporal fossa.

Histologic examination of the nasal and sinus tissue reveals large sectors of necrotic epithelium and submucosa. These necrotic areas have poorly defined cell outlines and a characteristic smudged appearance (fig. 1). The viable adjacent tissues contain intact segments of pseudostratified respiratory epithelium and an intense inflammatory response with congestion, edema, and neutrophilic infiltration (fig. 2). Other areas depict a chronic process. These show lymphocytes, plasma cells, giant cells, and proliferating fibroblasts (fig. 3). Similar areas of ulceration, necrosis, inflammation, and fibrous reaction involve the pharynx, larynx, and trachea.

The most common gross pulmonary lesions are dark red, rounded inflammatory masses.

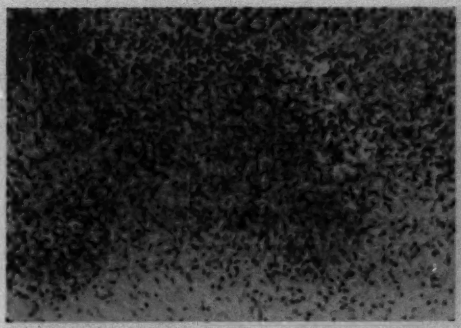


Fig. 3 (Straatsma). Maxillary sinus. Chronic inflammation and proliferating fibroblasts. (A26618. Hematoxylin-eosin, ×60.)



Fig. 5 (Straatsma). Lung. Necrosis with frank cavitation associated with a ragged outline and gray-green discoloration. Contiguous ocular involvement. (Case 3.)

These masses, measuring two to eight cm. in diameter, are sharply circumscribed (fig. 4). Necrosis in the central portion leads to softening or even frank cavitation associated with a ragged outline and gray-green discoloration (fig. 5). There seems to be a frankly remittent course in these lesions so that necrosis and reparative fibrosis alternate in the ascendancy. Distortion of pulmonary structure and bronchostenosis may develop as a late result of fibrosis and contracture.

Microscopic study of the pulmonary lesions reveals broad areas in which the alveolar structure and smaller bronchioles have been obliterated by necrosis and inflammation. The mucosa and submucosa of larger bronchi are disrupted (fig. 6) and blood vessels within the confines of this virulent process are infiltrated (fig. 7).

Surrounding the areas of necrosis are zones of inflamed granulation tissue. This contains the fibroblasts and blood capillaries of granulation tissue with the round-cell, epithelioid-cell, and giant-cell reaction of chronic inflammation. The term granuloma applies to this mass of inflamed granulation tissue[19] and the term necrotizing granuloma describes the particular pathologic situation in this tissue.

The second characteristic lesion of Wegener's granulomatosis is diffuse focal vasculitis involving the smaller arteries and, to some extent, the smaller veins. Sequelae in the form of aneurysm, hemorrhage, and thrombosis are not uncommon. Organs such

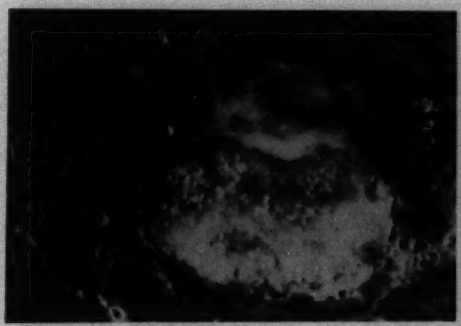


Fig. 4 (Straatsma). Lung. Sharply circumscribed, rounded inflammatory mass. Contiguous ocular involvement. (Case 6.)

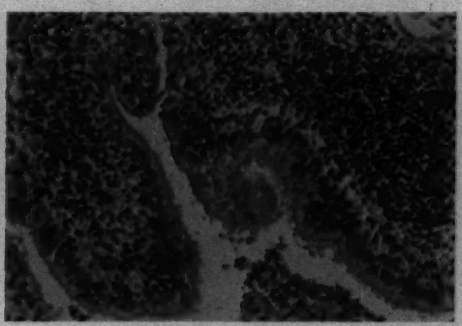


Fig. 6 (Straatsma). Lung. Inflammation and necrosis involving a bronchiole. (A23290. Hematoxylin-eosin, ×100.)

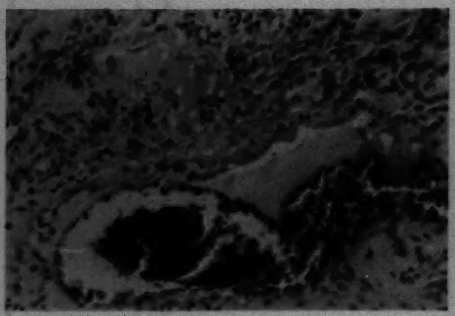


Fig. 7 (Straatsma). Lung. Inflammation involving a small vessel within the confines of the granulomatous process. (A23290. Hematoxylin-eosin, ×100.)

as the spleen, heart, pancreas, lung, and adrenal are subject to arteritis and infarction.[4,5]

Degeneration and infiltration characterize the acute arteritis (fig. 8). Other lesions seem to be older and contain evidence of fibrosis and healing. The evolution of arterial lesions through the stages of degeneration, inflammation, fibrosis, and recanalization is also noted in periarteritis nodosa. This sequence emphasizes the recuperative powers of the arterial system and has important therapeutic implications.

Renal lesions, the third characteristic anatomic alteration, are directly responsible for the fatal outcome in most cases. Usually the kidneys are moderately enlarged and contain numerous punctate hemorrhages. In



Fig. 9 (Straatsma). Kidney. Large hemorrhagic foci and yellow wedge-shaped infarcts. Contiguous ocular involvement. (Case 3.)

other cases there are larger hemorrhagic foci and a variable number of yellow wedge-shaped infarcts (figs. 9 and 10). These gross tissue alterations are related to fibrinoid necrosis, inflammation, and occlusion of the medium and small renal arteries. However, the characteristic microscopic lesion is focal necrotizing glomerulitis. This process entails necrosis and thrombosis of capillary lobules in the renal glomerulus.

Ophthalmic aspects

Following this introductory review of Wegener's granulomatosis, attention may be given to the ophthalmic aspects of this syndrome. Positive ocular findings related to Wegener's disease are specifically noted in

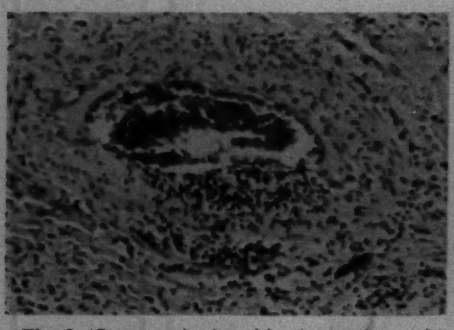


Fig. 8 (Straatsma). Arteritis. Acute phase with degeneration and inflammatory cell infiltration. (A23290. Hematoxylin-eosin, ×100.)



Fig. 10 (Straatsma). Kidney. Prominent streaked hemorrhages and pale yellow infarcts. Contiguous ocular involvement. (Case 3.)

TABLE 2

OCULAR MANIFESTATIONS OF WEGENER'S GRANULOMATOSIS

Case	Reference	Age	Sex	
	Contiguous Involvement			
1	20	49	M	
2	4, 5	34	M	
3	4, 5*	39	M	
4	1	70	M	
5	21	21	F	
6	4, 5*	38	M	
	Focal Involvement			
1	24	67	F	(retina)
2	21	21	F	(uvea)
3	26	35	F	(uvea)
4	8, 9	12	M	(uvea)
5	27	27	M	(conjunctiva)
6	4, 5	12	F	(conjunctiva)
7	12	39	M	(conjunctiva)
8	10	41	F	(conjunctiva)
9	13	24	F	(conjunctiva)
10	28	41	F	(corneosclera)
11	8	45	F	(corneosclera)
12	11	42	F	(corneosclera)
13	12	34	F	(corneosclera)
14	14	42	M	(corneosclera)

* Hospital record and protocol received.

19 of the 44 documented cases (table 2). The kaleidoscope of lethal disease diverts attention from the visual system so that this record of ocular involvement in almost half of the patients undoubtedly represents a minimum estimate. Nonetheless, ocular involvement was the first and most persistent sign of disease in at least one patient[8] and visual failure with ultimate blindness posed the primary clinical problem in another.[4]

The ocular involvement in Wegener's granulomatosis may be conveniently divided into contiguous and focal. The contiguous form applies to orbital disease that is a direct spread from adjacent diseased respiratory passages. The term focal is reserved for discrete areas of involvement lacking continuity. Quite obviously this division is arbitrary, but some classification is essential for orderly consideration.

CASE REPORTS

Contiguous involvement of varying degree was noted in six reported cases. These patients will be considered briefly with emphasis on the ocular alterations.

Case 1. This 49-year-old man presented with nasal obstruction and facial pain. Examination revealed raw ulcerated nasal mucosa. Two months later he developed slight left exophthalmos and X rays showed inflammation of the left antrum. This exophthalmos persisted during the remainder of the patient's life.[20]

Case 2. Bilateral proptosis and conjunctival congestion developed in this 34-year-old man, with pansinusitis, migratory arthritis, and urinary casts. Exophthalmos persisted during his six-month downhill course.[4, 6]

Case 3. A 39-year-old man was hospitalized with the complaint of facial pain and nasal discharge. Examination disclosed left exophthalmos, bone destruction in the floor of the orbit, and a granulomatous mass in the left antrum. Left exophthalmos increased to seven mm. and was associated with exposure keratitis, chemosis, limitation of ocular motility, and congestion of fundus veins. Death occurred three months after hospital admission. Autopsy confirmed the bony defect in the orbital floor and the direct communication between the left orbit and the aggressive granulomatous process in the left antrum.[4, 6]

Case 4. Following almost a year of respiratory tract signs and symptoms, this 70-year-old man developed bilateral asymmetrical exophthalmos and vision decreased to near blindness. Death occurred two weeks later. Autopsy disclosed clouding and bulging of the dura mater in the area of the optic nerves. Beneath this inflamed dura was a foul smelling discolored granuloma that had destroyed the roof and the anterior wall of the sphenoid sinus.[1]

Case 5. A 21-year-old woman noted nasal obstruction and fever prior to the onset of decreased vision and pain in the right eye. Vision in this eye decreased to counting fingers. Visual fields revealed a central scotoma. This was combined with right exophthalmos, lid edema, ptosis, restricted ocular motility, and impaired pupil response to light. Post-mortem examination disclosed a partially necrotic organized granuloma encasing the optic nerve. The inflammatory reaction was confined to the small vessels of the meninges and the nerve itself appeared normal.[21]

Case 6. A 38-year-old man was hospitalized with fever and pleuritic chest pain. Positive findings were sinusitis and a mass in the left lung. Left lower lobe pulmonary resection and sinus biopsy formulated the diagnosis of Wegener's granulomatosis. Despite antibiotics and bilateral Caldwell-Luc procedures, severe destructive pansinusitis, and rhinitis progressed. Left exophthalmos developed with recurrent chemosis, limitation of motility, papillitis, impaired pupillary response, and complete loss of vision. Several months later the other eye developed exophthalmos, chemosis, limitation of motility, and papilledema, although vision remained good for about one year. During this period treatment included X-radiation, nitrogen mustard, triethylene melamine, stilbestrol, testosterone, and snake venom. However, ACTH and cortisone re-



Fig. 11 (Straatsma). Excavated nasal cavity. The cranial cap and brain have been removed and the skull sectioned sagittally. This view demonstrates a completely excavated nasal cavity extending from the base of the skull to the tongue. Lining this cavity is a shaggy, firm, gray-red membrane composed of granulation tissue with varying degrees of necrosis. Contiguous ocular involvement. (Case 6.)

duced the intensity of inflammation, pain, and fever more effectively than other measures. After nearly two years of steroid therapy, extreme edema forced cessation of this treatment and all vision was lost. Some vision returned to the right eye when ACTH was reinstituted, but this slowly failed. The patient was totally blind when death occurred 42 months after onset of the illness.

Autopsy demonstrated a completely excavated cavity extending from the base of the skull to the tongue. This was lined by a shaggy, firm, gray-red membrane composed of granulation tissue with varying degrees of necrosis (fig. 11). Similar shaggy gray tissue extended into the maxillary, ethmoid, and sphenoid sinuses. The membrane was tightly adherent to the wall of the ethmoid sinus and eroded the bone to pass into the cranial cavity and apparently infiltrate about the optic nerves. The optic nerves, chiasm, and optic tracts were reduced in size. Microscopically, these structures showed loss of myelin and diffuse astrocytosis.[4, 8]

Discussion

Appraisal of these six cases delineates a distinct pattern of contiguous orbital involvement. Every case had antecedent or coincident disease in an adjacent portion of the upper respiratory tract. Extension of this process to the orbit occurred in the absence of a grossly demonstrable communication or through defects in the boundaries of the maxillary, sphenoid, or ethmoid sinuses. Exophthalmos was the primary sign of orbital encroachment. Other signs present in varying number and fluctuating in intensity were exposure keratitis, chemosis, limitation of motility, congestion of fundus veins, and papilledema. Impairment of vision was probably secondary to increased orbital pressure. The deleterious effect of this pressure on the optic nerve, particularly the maculopapular bundle, is somewhat analagous to the decrease in vision and central scotoma noted in endocrine exophthalmos and pseudotumor. Therapeutically significant is the fact that this process is generally reversible if orbital pressure is promptly controlled.[22, 23]

The second major form of ocular involvement in Wegener's granulomatosis is focal disease. Ocular involvement that is distinct and separate from respiratory tract pathology is not unexpected in this disseminated syndrome and was present in 14 patients. (One case[21] had histologically proven contiguous and focal disease.)

In one patient, narrowing of the retinal arterioles and cotton-wool patches were noted in the absence of hypertension.[24] These cotton-wool patches are generally considered synonymous with cytoid bodies and have been shown by Friedenwald[25] to represent small retinal infarcts that occur with occlusive vascular disease.

Uveitis without distinctive characteristics was present in three patients.[5, 9, 21, 26] Histologic confirmation of uveal inflammation was obtained in two cases[5, 9, 21] and both showed a nonspecific vasculitis and granulomatosis.

Five patients had conjunctival inflammation in association with skin or mucous membrane eruptions.[4, 5, 10, 12, 13, 27] The conjunctival lesions were not described in detail, but the cutaneous reactions were generally hemorrhagic, vesicular, and bullous (fig. 12).

The most significant focal ocular involvement—present in five patients—was inflammation of the sclera and cornea. Three cases had nonspecific episcleritis and scleritis.[11, 12, 14] Two other patients exhibited a paralimbal corneal infiltration with subse-



Fig. 12 (Straatsma). Skin reaction. Hemorrhagic and bullous cutaneous eruption. Contiguous ocular involvement. (Case 6.)

quent ulceration and formation of a furrow.[8, 28]

This corneoscleral involvement combines with conjunctival, uveal, and retinal implication to emphasize the diverse clinical manifestations that may stem from focal ocular involvement. It is of clinical interest that these focal manifestations are similar to lesions described in periarteritis nodosa. The close relationship between Wegener's granulomatosis and a number of diseases makes mandatory a consideration of differential diagnosis. The most detailed description of general anatomic alterations and specific ocular involvement is of no value unless a useful frame of reference is established. No attempt will be made to list all the clinical and pathologic conditions that may mimic various phases of this syndrome. Rather, an effort will be made to show that this entity lies in the middle of a broad compass of perplexing conditions. This spectrum ranges from purely vascular disease through mixed forms to purely granulomatous diseases.

The disseminated vascular lesions have led many authors to consider Wegener's syndrome a type of periarteritis nodosa. In this regard it is well to note that periarteritis nodosa is a collective term embracing at least two subtypes. One type attacks medium sized arteries, produces arterial aneurysms, inflammatory nodules, vascular obstruction, and infarction. The second form is a microscopic angiitis that involves smaller vessels.[29, 30] Neither form can account for massive necrosis and inflammation concentrated in the respiratory tract. Furthermore, disseminated granulomas distinct from vascular inflammation occur in Wegener's syndrome and are not consistent with any purely vascular disease.

Diffuse arteritis and granulomatous pulmonary inflammation occur in a group of cases histologically closely akin to Wegener's syndrome. These lesions have been found in patients who succumbed following intractable asthma, fever, and hypereosinophilia.[31, 32] The overt allergic manifestations and hypereosinophilia differentiate this group from Wegener's syndrome.

Purely granulomatous lesions are the characteristic tissue response in a wide variety of disease forms. Specific granulomatous diseases such as tuberculosis, syphilis, and histoplasmosis may be differentiated by identification of the etiologic agent. Boeck's sarcoid with diffuse pulmonary involvement and vascular lesions may clinically simulate Wegener's syndrome. However, the lesions of sarcoid are free of necrosis and distinction is made on this basis.

Midline lethal granuloma (granuloma gangraenescens) is a mutilating disease of unknown etiology characterized by granulomatous inflammation and progressive destruction of the nose and facial area (figs. 13 and 14). Various micro-organisms have been implicated,[33] but most authors feel that these are secondary invaders and attribute the tissue loss to other factors. Exhaustion and debilitation dominate the terminal phase of this disease, and death may result from erosion of a large vessel and massive hemorrhage.[34, 35] Comparatively few of these fatal cases have been subjected to complete postmortem study. However, in most of these autopsied patients the disease was confined to the facial tissues. Occasional cases have vascular and renal[35, 36] or pulmonary lesions[37] that suggest a relationship between restricted facial granuloma and the generalized granuloma of Wegener.[4, 5, 18, 38] Despite these tran-



Fig. 13 (Straatsma). Midline lethal granuloma. (E. R. September 22, 1948.)

sitional forms, it seems clear that midline lethal granuloma does not inevitably or universally terminate in Wegener's granulomatosis so that a distinction between the two conditions is advisable on clinical and anatomic grounds.

Somewhat distinct from this spectrum of vascular and granulomatous inflammation is the group of neoplastic diseases that may mimic purely inflammatory conditions. Pathologists recognize that granulomatous forms of Hodgkin's disease, mycosis fungoides, and lymphosarcoma simulate purely inflammatory foci. Clinicians know the importance of considering neoplasm—primary, contiguous, or metastatic—in the differential of an obscure process.

The final condition to be considered in



Fig. 14 (Straatsma). Midline lethal granuloma. (E. R. March 22, 1949.) This patient is alive at the present time and without evidence of disseminated vascular or granulomatous lesions. The bacteriologic aspects of this case have been reported.[22]



Fig. 15 (Straatsma). Trophic ulceration. This facial ulceration developed following nerve section for trigeminal neuralgia. (M. C. August 28, 1956.)

differential diagnosis is the extensive trophic ulceration that may develop following nerve section for trigeminal neuralgia (fig. 15). This is included not because of its close relationship to Wegener's syndrome, but to emphasize the deficit of fundamental information concerning the physiologic etiology and pathogenesis of many conditions.

It is with full realization of this basic gap in knowledge that consideration is given to the possible pathogenesis of Wegener's granulomatosis. It must be asserted that the cause of this condition is unknown and admitted that this consideration borders closely on speculation. However, speculation is justified in view of the potential importance of understanding the pathogenesis of Wegener's syndrome. A logical concept of pathogenesis must account for the anatomic alterations of granuloma, arteritis, and nephritis and endeavor to determine a common factor responsible for these lesions.

Granulomas are produced by a variety of agents. However, in most instances, this tissue response can be considered a foreign body reaction produced by an unabsorbable agent[39, 40] or a sensitivity reaction incited by an organism or allergen.

Diffuse focal arteritis is also related to a number of clinical and experimental situations including renal ischemia, precipitous blood pressure fluctuations, and hypersensitivity reactions.[29, 41, 42]

The role of hypersensitivity is even more

apparent in the production of glomerulonephritis. The antigen-antibody response has been repeatedly implicated both clinically and experimentally in the production of this form of renal disorder.[42]

It is thus apparent that allergy or hypersensitivity used in the general sense to mean altered tissue reactivity is capable of producing each and every significant anatomic alteration in Wegener's granulomatosis. Reinforced by this knowledge, it is possible to postulate a sequence of events consistent with the clinical course of Wegener's disease and terminating in the tissue alterations present at the death of these patients.

Initially, an unknown allergen or sensitizing substance comes into contact with the tissues of the respiratory tract. These tissues are particularly responsive to sensitizing agents whether micro-organism, pollen, or chemical, and react with an inflammatory response designed to localize the offending material. This localizing reaction is an important defensive mechanism exhibited by most tissues and is generally adequate to fix the allergen to the tissues until it can be destroyed by proteolysis. However, if the amount of allergen is excessive, it exceeds the capacity of the tissues and necrosis occurs. Repeated contact with the same allergen intensifies the inflammation and necrosis. Moreover, if the allergen gains access to the circulation, it is drawn to the site of inflammation and compounds or flares up the reaction in progress. This sequence of allergen localization, inflammation, and necrosis repeated over a long period may account for the remittent, yet relentless inflammation and destruction characteristic of the respiratory tract lesions in Wegener's syndrome.[43]

Somewhat later in the course of the disease, diffuse focal arteritis and nephritis develop as an allergic response to the same or different circulating allergens. Conceivably, the proteins released from the respiratory tract lesions may act as sensitizing substances.[4, 5] The tissue damage resulting from this generalized granulomatosis and vasculitis is greater than the organism can sustain, and death ensues.

This fatal syndrome—Wegener's granulomatosis—may then be considered as the full-blown response of the organism to an overwhelming allergic disorder.[38] However, in inflammatory and allergic disorders, mild as well as severe and focal as well as generalized disease is encountered. Experimentally, immunologic conditions can be varied to produce focal as well as generalized arteritis,[42, 44] and the severity of the granulomatous response can be controlled. A variety of clinical conditions within the realm of ophthalmology may be tentatively considered as examples of restricted or focal allergic disorders. The sequence of events undoubtedly differs in these focal allergic disorders although a factor of allergy or altered tissue reactivity is highly probable. The vascular inflammation in temporal arteritis and the focal retinal arteritis described by Klien[45] as Jensen's juxtapapillary retinopathy are two possible examples of focal vascular allergy. Lethal midline granuloma and some cases of orbital granuloma or pseudotumor may represent focal forms of granulomatous allergic response. The histologic appearance of pseudotumor with nonspecific inflammation, vasculitis, and fibrosis is consistent with this concept[46] (figs. 16 and 17).

Despite the speculative nature of this discussion concerning pathogenesis, clinical and experimental evidence support this concept.

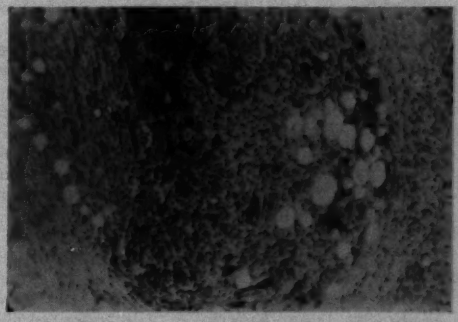


Fig. 16 (Straatsma). Orbital granuloma. Nonspecific inflammation, vasculitis and fibrosis. (I.O.P.H. 6140. Hematoxylin-eosin, ×30.)

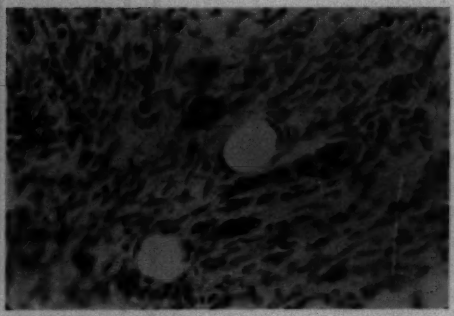


Fig. 17 (Straatsma). Orbital granuloma. Giant cells and round cells of chronic inflammation. (I.O.P.H. 6140. Hematoxylin-eosin, ×100.)

Only further studies into the basic causes of the tissue reaction can confirm or refute the theory of altered tissue reactivity as a pathogenetic factor in Wegener's granulomatosis and allied focal disorders. The immediate practical importance of this concept is to enlarge the scope of possible etiologic agents to include all allergens as well as the customary pathogenic micro-organisms. Moreover, treatment must be designed to control an overzealous sensitivity response as well as eliminate every possible etiologic agent.

Summary

Wegener's granulomatosis is a generalized fatal disorder characterized anatomically by necrotizing granulomatous lesions in the respiratory tract, diffuse arteritis, and focal glomerulonephritis. Ocular involvement of varying severity is present in almost half of the cases showing this triad. This ocular involvement may be contiguous extension from the respiratory system or discrete focal lesions affecting retina, uvea, conjunctiva, or corneosclera. Differential diagnosis includes a broad spectrum of diseases extending from purely vascular to purely granulomatous lesions. The etiology of Wegener's granulomatosis is unknown. However, a concept of pathogenesis is presented and this concept is related tentatively to other obscure ophthalmic conditions.

635 West 165th Street (32).

I wish to thank Dr. Algernon B. Reese and Dr. Gabriel Godman for assistance during the course of this study. I am also grateful to Dr. S. Otani and Dr. L. Strauss of the Mount Sinai Hospital, New York, for permission to use contiguous involvement—Case 3, and Dr. B. Gordon and Dr. T. Ehrenreich of the Veterans' Administration Hospital, Bronx, for permission to use contiguous involvement—Case 6.

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AN ANATOMIC STUDY OF THE CHECK MECHANISM OF THE VERTICAL MUSCLES OF THE EYES*

WALTER H. FINK, M.D.
Minneapolis, Minnesota

Although our knowledge of the check mechanism of the horizontal muscles is fairly well established, very little positive evidence is available as to the check mechanism associated with the vertical muscles. This is understandable when one considers the greater complexity in the anatomic arrangement of the vertical muscles as compared with the horizontal muscles.

With the hope of obtaining a clearer concept of the check mechanism of the vertical muscles, I have assembled some anatomic data pertaining to this subject. It is obviously not feasible to present a complete description of the check mechanism based on an anatomic study only. However, such a study may serve as a basis for further work, which will lead to a better understanding of this complex problem.

The investigation is based on the study of a series of chemically prepared anatomic specimens, and supplemented by the examination of living tissues of patients requiring an enucleation or muscle surgery. The living tissue presents a more accurate picture of the anatomy than can be found in the laboratory specimens.

For the purpose of presentation, the subject will be divided into two parts: first, the anatomic structures involved, and second, the role of these structures in the check action of the vertical muscles.

ANATOMIC STRUCTURES INVOLVED

The understanding of the check action of a vertical muscle necessitates a clear concept of the anatomy of the area. The following is a review of the anatomy of the fascial membranes associated with the various vertical muscles. A familiarity with the anatomy of the vertical muscles is assumed.

FASCIAL MEMBRANES OF LEVATOR

Although the levator muscle, strictly speaking, is outside the scope of this paper, its close association in the check action of the vertical extrinsic muscles necessitates its inclusion in an anatomic description of the area.

The posterior half of the levator muscle, for the most part, is devoid of a condensed membrane, being covered by only a thin mesh of fibro-elastic tissue which binds the muscle fibers to the surrounding reticulum of fatty tissue. In fact, this membrane over the posterior half of the muscle is so thin that the muscle fibers appear practically devoid of covering. Anteriorly, however, the fascial sheath becomes gradually thicker and more evident until, just posterior to the aponeurosis, the whole muscle is enveloped in a thick, opaque sheath (fig. 1). This sheath on the upper aspect of the muscle becomes progressively more dense anteriorly and, just posterior to the orbital rim, becomes condensed into a quite definite ligamentous band which stretches transversely across to reach the orbital walls on either side. It extends parallel with the aponeurosis of the levator, being inserted into the bony orbit at a slightly higher level. In front of this ligamentous condensation, the sheath becomes abruptly so thin that the thickened band appears to end in a free border; however, the thin membrane can be traced forward and up to the supraorbital border, where it blends with the orbital septum.

Laterally, the expansion of the sheath follows the course of the aponeurosis and, anteriorly, divides the lacrimal gland into

* Presented at the 92nd annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, June, 1956.

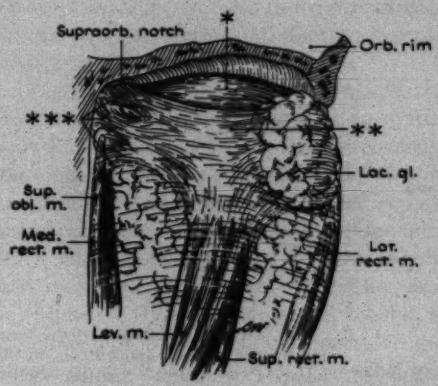


Fig. 1 (Fink). Fascial sheath of the levator muscle and its relation to the adjacent structures, viewed from above. (* Extension of the fascial sheath to the orbital rim. ** Fascial sheath of the levator muscle. *** Medial insertions of the fascial sheath of the levator muscle.)

two portions. Above, it blends with the fascial covering of the lacrimal gland, resulting in a firm union between the sheath and gland, and, in addition, blends with the surrounding fatty reticulum. Beneath, it blends with the aponeurosis of the levator and the lateral expansion of the sheath of the superior rectus muscle.

Medially, the sheath of the levator is fused mainly to the trochlea. In addition, it sends bands forward to the periosteum of the orbital rim, where it bridges the supraorbital notch. Posterior to the trochlea, the membrane becomes fused to the periorbita of the bone in the region of the superior oblique muscle just before it enters the trochlea. Above, it is fused with the reticulum of fibro-elastic tissue which contains the orbital fat, and is thus indirectly connected to the periorbita covering the roof of the orbit. Beneath, it is fused to the medial expansion of the aponeurosis of the levator, accompanying it to its insertion. In addition, it blends with the medial expansion of the fascial sheath of the superior rectus.

The fascial sheath covering the inferior surface of the levator muscle fuses with the fascial sheath of the superior rectus muscle, creating a union between the two (fig. 2). A fascial structure is thus formed which fills up the angle of divergence between the muscles as they pass to their respective insertions—one to the lid, and the other to the globe. Anteriorly, this fascial structure is fixed to the conjunctiva along its line of reflection from the lid to the globe (the superior fornix). Although no muscle fibers pass into it from the levator, the attachment is described as an additional insertion of that muscle. It is by means of this fascial attachment that the conjunctiva is drawn up and backward when the globe is rotated upward.

Fascial membranes of superior rectus

As in the case of the levator muscle, the posterior half of the superior rectus muscle is practically devoid of a membranous sheath. Except for its medial border, it is covered by a loose, meshlike, fibro-elastic membrane which is closely adherent to the surrounding reticulum of fatty tissue. On separating the levator muscle from the superior rectus, one finds a very definite fibro-elastic band along the entire medial border of the superior rectus, extending to the inner border of the levator muscle above. The medial edges of the two muscles are thus firmly bound together in the posterior half. In the anterior half, however, this fascial band gradually expands laterally, and eventually completely covers the opposing surfaces of the muscles, which become completely fused. This fusion has been described previously in connection with the levator muscle fascia. Although fixed tissues give the impression of a firm fusion, the examination of living tissues shows it to be sufficiently loose and elastic to permit the necessary independent movements of the lid and eyeball.

It is important to point out that the fusion between the two fascial sheaths is not limited to a few millimeters anteriorly, but extends posteriorly, laterally, and medially, involving the whole area of the fascial coverings and their expansions.

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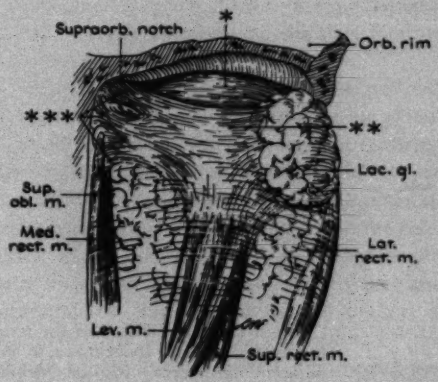


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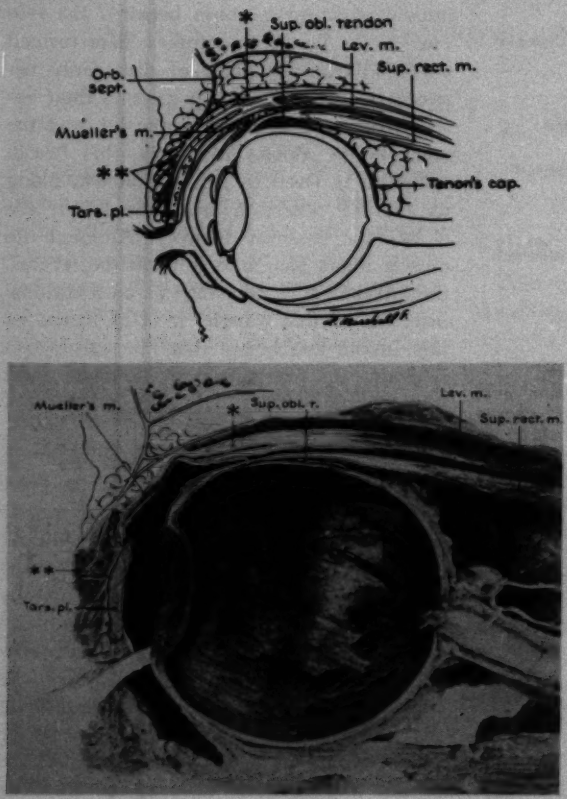


Fig. 2 (Fink). Relation of the levator muscle and its aponeurosis to the adjacent structures. Sagittal section through midplane of the eyeball. (*Palpebral aponeurosis. ** Terminal fibers of the levator which pass through the orbicularis muscle to terminate in the skin of the lid.)

In the anterior half of the superior rectus muscle, the fascia not only completely encircles the muscle, but in addition expands both laterally and medially to fuse with adjacent structures. The lateral expansion becomes fused with the fascial expansion of the lateral rectus and, with the fascial covering of the levator, follows a lateral course, and eventually becomes inserted in the periorbita at the outer angle. It likewise passes through the anterior portion of the lacrimal gland.

Medially, the expansion of the superior rectus fuses with the fascial covering of the medial rectus. Above, it fuses with the fascia of the aponeurosis. Below, it fuses with the expansion of the fascia bulbi which covers the tendon of the superior oblique. It is inserted into the trochlea, the periorbita adjacent to it, and a portion of it terminates with the insertion of the aponeurosis and its fascia.

In addition to the above, the fascia of the superior rectus fuses with the fatty reticulum which surrounds it, and is thus indirectly connected to the periorbita. The fascia covering the under surface of the muscle extends anteriorly to the point of insertion of the muscle and is in direct contact with the fascia bulbi which surrounds the eyeball.

Fascial membranes of superior oblique reflected tendon

The fascial membranes covering the reflected tendon may be divided into two parts:

the part from the trochlea to the medial edge of the superior rectus, and the part beneath the superior rectus.

The first part of the sheath of the reflected tendon of the superior oblique is an extension of various fascial membranes covering the muscles and the fascia bulbi to the trochlea and adjacent areas. The various membranes form a sheath two to three mm. thick about the tendon, so that the tendon and its sheath together are five to six mm. in diameter. The sheath of the tendon is comprised of the medial expansion of the sheath of the levator muscle, the medial expansion of the sheath of the superior rectus muscle, and the extension of the fascia bulbi. The tendon of the superior oblique is united to its fascial sheath by numerous fine fibrillas which permit a limited independent movement between the sheath and the tendon (fig. 3). This sheath completely encloses the trochlea, from which extensions can be seen to pass to the orbital septum in front, the muscle fibers of the superior oblique behind, and the periorbita adjacent to it.

The second portion of the superior oblique tendon covering, which is beneath the superior rectus, has a somewhat less complicated arrangement. It is covered by the fascia bulbi and the other structures before described. Trabeculas surround the fan-shaped expansion of the tendon as it approaches the point of insertion, binding it to the fascia bulbi above and the sclera below. The trabeculas in this region consist of broader bands of fibro-elastic membrane rather than tiny hairlike fibers; because they are of this type, the movements of the globe are not restricted (fig. 4).

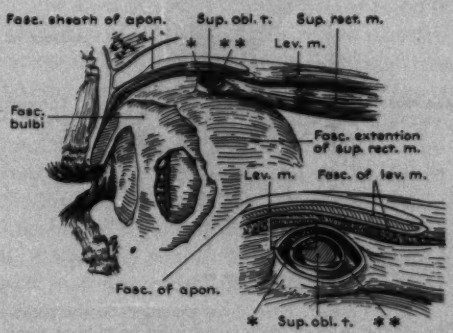


Fig. 3 (Fink). Fascial covering of the superior oblique tendon. Sagittal section at the plane of insertion of the medial rectus muscle. (* Medial extension of the fascial coverings of the superior rectus muscle which blends with the condensation of the surrounding reticulum. ** Extension of the fascia bulbi.)

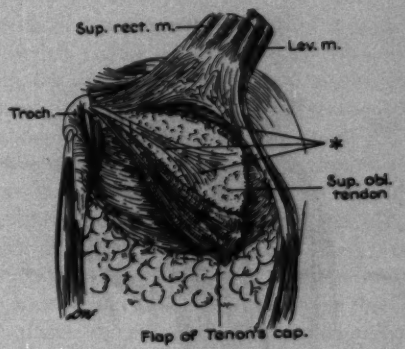


Fig. 4 (Fink). Relation of the fascial sheath to the superior oblique tendon, viewed from above. The fascial covering has been incised and a triangular flap reflected backward, showing the tendon of the superior oblique beneath. The fascial sheath of the superior oblique tendon is anchored in place on the medial end by its fusion to the trochlea and the surrounding area. On its lateral end it is continuous with the fascial coverings of the levator, superior rectus, and the fascia bulbi. Arising from the inferior surface of the fascia bulbi are many fibro-elastic bands, or trabeculas, which form a union between it and the oblique tendon. (* Cut ends of trabeculas which extend from the sclera to the fascia bulbi.)

Fascial membranes of inferior oblique

Fascial membrane encloses the inferior oblique from its periorbital origin to its insertion and extends to the adjacent structures, fusing with their fascial coverings.

The fascia in this area, usually referred to as the suspensory ligament of Lockwood, consists of a blending of the sheaths of the inferior oblique and inferior rectus muscles to form a special thickening of the fascial structure. The expansions of this sheath

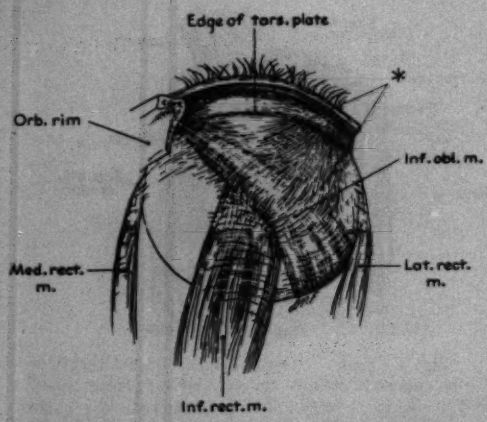


Fig. 5 (Fink). Fascial membrane covering the inferior oblique and inferior rectus muscles, viewed from below. The fascial covering of the inferior oblique muscle continues forward to fuse with the tarsal plate and the orbital septum. The fascial connection thus formed between the two areas is more compact near the point where the two muscles cross. (* The area at the crossing of the inferior rectus and the inferior oblique muscles, where the membrane is more dense.)

extend upward laterally and medially to join the sheaths of the lateral and medial rectus muscles, thereby gaining indirect attachment to the orbital margin. There is thus formed a continuous band, about one tenth of an inch thick, beneath the globe, supporting it like a hammock (fig. 5).

Lockwood described the fascial arrangement as follows:

> The suspensory ligament is a band of fibrous tissue stretched, like a sling, from one side of the orbit to the other. The fibers which compose it converge at each end to be inserted into the malar and lacrimal bones; in the middle they diverge to form a shallow cup upon which the eye rests. The widest part of the suspensory ligament is intimately woven with the capsule of Tenon, but not to such an extent as to conceal the identity of its fibers.

Anatomic material reveals a seemingly firm union between the inferior oblique and the inferior rectus muscles, produced by the fusion of the fascial sheaths of these muscles, at the point of crossing. The fascial sheath on the undersurface of the inferior rectus muscle becomes definitely thickened a few millimeters before it reaches the inferior oblique, and, at the point where the muscles cross, this thickened fascia comes in contact with a similar thickening of the fascia covering the inferior oblique. A fusion is brought about by numerous fascial bands passing from one fascial sheath to the other (fig. 6).

The fascial bands seem to terminate anteriorly in the adjacent orbital septum and the orbital rim. Some strands from the inferior portion of the fascial covering of the oblique seem to blend with the supporting structure of the fatty tissue and are thus indirectly attached to the periorbita on the floor of the orbit immediately behind the orbital rim.

As in the upper lid, where the levator palpebrae and the rectus superior are united by fibro-elastic tissue which is attached to the upper cul-de-sac, in the lower lid an extension of the ligament of Lockwood is attached to the lower tarsal plate and cul-de-sac and tends to hold them in place. It also sends fascicular bands to the skin and intermuscular tissue of the lower lid (fig. 7).

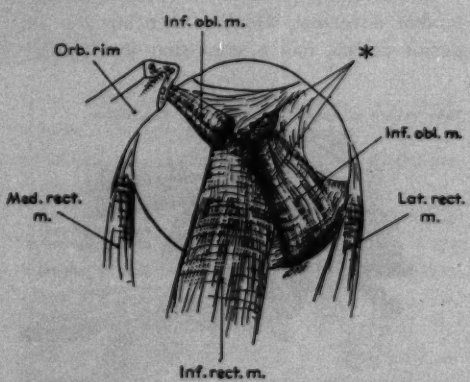


Fig. 6 (Fink). Fascial arrangement at the crossing of the inferior rectus and inferior oblique muscles, viewed from below. In the drawing the inferior oblique muscle is severed and turned laterally, showing a union between the fascial sheaths of the two muscles. Bands of fibro-elastic tissue at the crossing of the muscles result in a fusion between the muscle sheaths. (* Fibrous bands between the inferior rectus and inferior oblique muscles.)

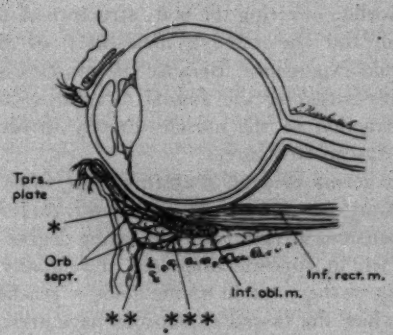


Fig. 7 (Fink). Fascial arrangement at the crossing of the inferior rectus and inferior oblique muscles. Sagittal section through midplane of eyeball. The fascia in this area is part of the suspensory ligament of Lockwood. An important feature is the fusion of the sheaths of the inferior oblique and inferior rectus muscles. The fascial membranes extend forward to the lower fornix, to the lower tarsal plate, and to the orbital septum. A few strands extend to the periorbita on the floor of the orbit. The latter are usually not direct extensions, but are made up of the fibrous meshwork which surrounds the fatty tissue. The extensions of the ligament of Lockwood, which are attached to the lower tarsal plate and fornix, tend to hold these structures in place. Fascial extensions are also found in the skin and intermuscular tissue of the lower lid. (* Fibrous bands extending to the tarsal plate. ** Fibrous bands extending forward to the orbital septum. *** Fibrous bands extending to the periorbita on the floor of the orbit.)

Fascial membranes of inferior rectus

The sheath of the inferior rectus is quite similar to that of the other rectus muscles, except anteriorly, where it divides into two layers; a superior one which joins the fascia bulbi and runs forward to become inserted into the sclera near the limbus, and an inferior layer which is fused to the sheath of the inferior oblique at the point of crossing. The fascial sheath of the inferior rectus and its medial and lateral fascial expansions take part in forming the suspensory ligament of Lockwood.

Role of anatomic structures in check action of vertical muscles

General considerations

In considering the fascial check mechanism of the vertical muscle of the eye, we are not dealing with only a localized structure. Although the structures directly associated with the individual muscle may be primary factors in checking the action of the muscle, it should be emphasized that the check action on the muscle involves the entire orbital contents to greater or less degree.

Although all the extrinsic muscles act to check the action of a vertical muscle, the homolateral direct antagonist of the contracting muscle plays the most important role. If the muscle tonus is normal, the antagonistic muscle will exert a check action on the contracting muscle when the rotation of the globe reaches a point at which further relaxation of the antagonist ceases.

As in the case of the horizontal muscles, all of the orbital fascial structures are involved in the check action of a contracting vertical muscle, some of which play a more important role than others. The fascial structures which are chiefly responsible for check action of a vertical muscle are those directly associated with the vertical muscles. For the purpose of presentation they may be divided into two groups—the fascia associated with the vertical muscles above the globe (superior rectus and superior oblique) and the fascia associated with the vertical muscles below the globe (inferior rectus and inferior oblique).

A study of the anatomy of the area shows that the fascial sheaths and their expansions associated with the superior rectus and superior oblique tendon are fused together to form a continuous structure extending from the midpoint of the outer orbital rim to the midpoint of the medial orbital rim. In addition, this fascial structure is indirectly connected with the periorbita of the upper rim and roof of the orbit by the fibro-elastic reticulum which surrounds it. The fascial structure which is formed by this fusion of the various fascial membranes can be likened to a transverse band extending across the orbit, and may be referred to as the superior transverse fascial expansion. It is anchored securely in place and has a limited amount of mobility. Because of its fusion

with the various muscles in the area, it is directly involved in the various actions of these muscles.

Likewise the fascial membranes associated with the inferior rectus and inferior oblique form a transverse fascial expansion which may be referred to as the inferior transverse fascial expansion and is commonly known as the ligament of Lockwood. This structure is made up of the fused fascial expansions of the fascial sheaths of the inferior oblique and the inferior rectus. The fascial structure extends laterally and medially to fuse with the sheaths of the horizontal muscles and eventually becomes attached to the periorbita at the midpoint of the lateral and medial orbital rim. In addition it fuses with the periorbita of the lower orbital rim and through the surrounding fibro-elastic reticulum is attached to the periorbita covering the floor of the orbit.

Because of this anatomic arrangement, check action on the vertical muscles is possible. To demonstrate the effect of vertical movements of the globe on the transverse fascial expansions, a dissection of an orbit was made in which all structures were removed from the orbit except the eyeball, vertical muscles, and their associated fascia. A specimen was thus obtained in which the globe was suspended in the orbit by the superior and inferior fascial expansions. These, as above described, were made up of the fascial sheaths of the vertical muscles and their expansions.

It could be demonstrated that, when the globe was rotated downward, the superior transverse expansion moved forward and the inferior fascial expansion moved backward to an equal degree. The movement of the transverse expansions was reversed when the globe was rotated upward.

Although it was not possible to demonstrate accurately the extent of movement of the transverse expansions in a prepared specimen, it is logical to assume that in the living tissues there is a limit to the amount of movement of these structures. This is because of their extensive attachment to the periorbita covering the bony structure of the orbit and the relative inelasticity of the fascial expansions. Because the muscles are firmly fused to the fascial structures, any contraction of the muscle directly involves the fascial structures.

It seems evident, therefore, that when a vertical muscle contracts, the two transverse expansions are put to an increasing degree of stretch in opposite directions. Eventually a point in the rotation of the globe is reached, at which the fascial expansions have arrived at the limit of their movement and further rotation of the globe in that direction is stopped.

In addition to this check action, other factors of less importance are present, which serve to augment the check action of the transverse fascial expansions. The most important of these is the fusion of the muscle sheaths to Tenon's fascia. Tenon's fascia is comparatively immobile because of its fusion to the supporting reticulum of the orbit, which indirectly connects it to the periorbita.

Check mechanism of the superior rectus

Granting the action of various other fascial structures which may act to check the movement of the superior rectus, it is evident that an important check mechanism relates to the fascial fusion of the superior rectus with the levator. It is therefore necessary to include a description of the check mechanism of the levator in the discussion.

It is generally believed that the action of the levator is checked by the attachments of its fascial sheath. These attachments of the fascial sheath are the medial and lateral attachments to the periorbita at the midpoint of the rim of the orbit and to the periorbita of the roof of the orbit, made possible by the dense fibro-elastic reticulum which indirectly connects the two fascial structures. Through these fascial attachments the check action of the levator is achieved.

An important consideration in our present discussion is the fact that the fascial sheath covering the inferior surface of the levator

muscle fuses with the fascial sheath of the superior rectus muscle, creating a union between the two. A fascial structure is thus formed which fills up the angle of divergence between the muscles as they pass to the respective insertions—one to the lid, and the other to the globe. Anteriorly, this fascial fusion is fixed to the conjunctiva along its line of reflection from the lid to the globe (the superior fornix).

The fascia on the under surface of the levator, which fuses with the fascial covering of the superior rectus, creates a unified action between the levator and superior rectus and thus permits the fascial sheath of the levator to act as a check mechanism to both the levator and the superior rectus muscle.

This check action of the superior rectus muscle is described by Whitnall[2] as follows: "The sheath of the superior rectus blends with that of the overlying levator palpebrae superioris, and the fixed mass between them is attached to the back of the superior conjunctival fornix; the two muscles act harmoniously, and the superior rectus may be limited in range by the same fascial mechanism as has been described for the levator."

Although a study of the fascial fusion as seen in laboratory specimens gives the impression of a firm fusion between the two muscles, examination of the living tissues shows a certain degree of movement between the muscles thus permitting the necessary independent action of the lid and globe.

It is important to point out that the fusion between the two fascial sheaths is not limited to the fusion between the levator and the superior rectus, but includes a fusion between the fascial membranes which extend laterally and medially from the fascial covering of the two muscles to become inserted into the lateral and medial orbital rim at its midpoint. This structure, as previously mentioned, may be designated as the superior transverse fascial expansion.

In addition to the above fascial structure, the fascial membranes associated with its direct antagonist, the inferior rectus, are important in checking the action of the superior rectus. The fascial structures associated with the inferior rectus consist of a fusion of the fascial sheath of the inferior rectus and the sheath of the inferior oblique, along with their various fascial expansions. This structure, as described previously, may be designated as the inferior transverse fascial expansion or the ligament of Lockwood.

It is therefore evident that the check action of the superior rectus involves two major factors, namely, the fascial membranes associated with the superior rectus (superior transverse fascial expansion) and the fascial membranes associated with the inferior rectus (inferior transverse fascial expansion or ligament of Lockwood).

As the superior rectus contracts to move the globe upward, the superior transverse fascial expansion is put to a progressively greater backward stretch and eventually reaches its limit of backward movement. Simultaneously, the inferior transverse expansion moves forward and, if upward movement of the globe continues, the inferior transverse expansion moves forward and the inferior fascial extension likewise reaches a point where forward stretching of the structure ceases and checks further upward movement of the globe (fig. 8).

The fusion between the fascial sheath of the superior rectus and the fascia bulbi also exerts a check action on the superior rectus. The fascia bulbi, being attached to the surrounding fibro-elastic reticulum, is limited in its movement and thus, by its fusion to the under surface of the superior rectus, exerts a check action upon it.

Fascial check mechanism of superior oblique

The check mechanism of the superior oblique muscle has been the subject of considerable controversy. Because of the complex anatomy of the area and the variable actions of the superior oblique muscle, the check mechanism is not as evident as that found in the rectus muscles and consequently is more difficult to describe.

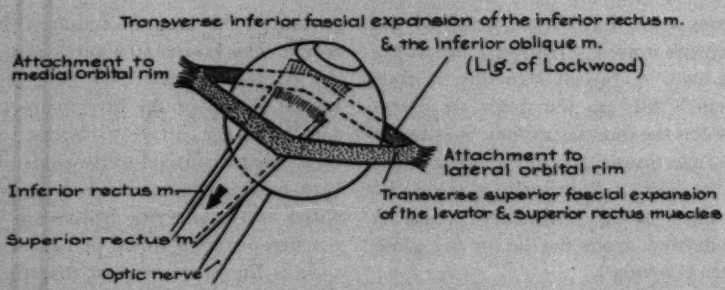


Fig. 8 (Fink). Diagram illustrating the position of the superior and inferior transverse fascial expansions when looking upward. The check action of the superior rectus involves two major factors, namely, the fascial membranes associated with the superior rectus (superior transverse fascial expansion) and the fascial membranes associated with the inferior rectus (inferior transverse fascial expansion or ligament of Lockwood).

As in the case of the superior rectus, two factors are chiefly responsible for its check action. A major factor in the check action on the superior oblique is the fascial covering of the superior oblique reflected tendon. This fascial covering of the superior oblique tendon, as described previously, consists of the medial expansion of the fascial sheath of the levator, superior rectus, and the fascia bulbi, which make up the superior transverse fascial expansion. Because the oblique tendon is attached to its fascial covering by innumerable fibrillae which permit only a limited independent movement of the tendon in its sheath, any action of the superior oblique tendon to rotate the globe will involve this fascial structure.

Another major factor in producing check action of the superior oblique muscle is dependent upon the fascial arrangement of its homolateral antagonist, the inferior oblique. This fascial structure, as previously mentioned, is referred to as the inferior transverse fascial expansion (ligament of Lockwood). Like the superior transverse fascial expansion, the inferior fascial structure is very stable in its position, because its extensions are anchored to the periorbita of the orbital rim laterally, medially and inferiorly. In addition, through the surrounding reticulum it is anchored to the periorbita on the floor of the orbit.

The movement of the globe in the field of action of the superior oblique causes a simultaneous movement of both the superior and inferior transverse expansions, the superior moving forward and the inferior backward. As these expansions are stable in their position, there is a limit to the amount of extension possible. When the superior oblique contracts, the fascial structures are increasingly stretched and eventually reach a point at which further movement of the fascial expansions is not possible and the action of the superior oblique is checked.

It therefore seems evident that the fascial membranes associated with its direct antagonist, the inferior oblique, and the fascial membranes associated with the superior oblique tendon are the major factors in limiting the rotation of the globe in the field of action of the superior oblique muscle.

Check mechanism of inferior oblique

A major factor in the check action of the inferior oblique is the fascia which immediately surrounds it. This fascial structure is referred to as the inferior transverse fascial expansion(ligament of Lockwood) and has been previously described.

Another important check action affecting the inferior oblique involves the fascial structures related to its homolateral antagonist, the superior oblique tendon. As previously mentioned, the fascial structures which are immediately associated with the superior oblique tendon are medial expansions of the fascia covering the levator,

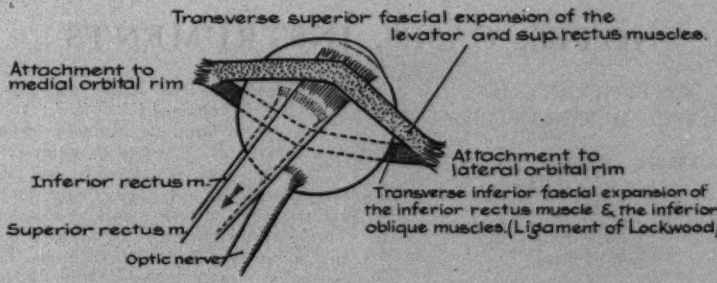


Fig. 9 (Fink). Diagram illustrating the position of the superior and inferior transverse fascial expansions when looking downward. The superior transverse fascial expansion acts in conjunction with the inferior transverse fascial expansion to check the action of the inferior rectus.

superior rectus, and the fascia bulbi, and make up the superior transverse fascial expansion.

When the inferior oblique muscle contracts to move the eye in its field of action, the superior and inferior transverse fascial expansions move simultaneously in opposite directions, the superior moving backward and the inferior moving forward. As the rotation of the globe continues, they gradually reach their limit of movement, and further rotation of the globe is stopped.

Check action is also exerted by the fusion of the fascial covering of the inferior oblique with the fascia bulbi.

Fascial check mechanism of inferior rectus

As in the case of the superior rectus where it is fused with the overlying levator, there is a somewhat comparable arrangement in the inferior rectus where it is fused with the inferior oblique at the point of crossing of the muscles. As previously described, the fascial expansions of the two inferior muscles are attached laterally and medially to the bony orbital rim at the midpoint to form the inferior transverse fascial expansion (ligament of Lockwood) and, as in the case of the inferior oblique, serve to check the action of the inferior rectus.

The second important factor in the check action of the inferior rectus is the fascial arrangement of its homolateral antagonist, the superior rectus. This structure, which has been previously referred to as the superior transverse fascial expansion, acts in conjunction with the inferior transverse fascial expansion to check the action of the inferior rectus. When the inferior rectus contracts to turn the globe downward, the transverse expansions move in opposite directions, the superior moving forward and the inferior moving backward. As the downward movement of the eye continues, the tension on the transverse expansion increases, and eventually the limit of their extensibility is reached and further movement of the globe is checked (fig. 9).

As in the case of the other vertical muscles, check action is also exerted by the fusion of the inferior rectus muscle sheath with the fascia bulbi.

Conclusion

The purpose of the presentation is to describe an anatomic basis for the check action of the vertical muscles. It is hoped that it will serve as a step toward a better understanding of this complex problem.

Medical Arts Building (2).

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NOTES, CASES, INSTRUMENTS

POSTVACCINIAL DISCIFORM KERATITIS*

Recurrent episodes treated with steroids

R. David Sudarsky, M.D.
New York

The occurrence of a disciform type of keratitis as a complication of vaccinia of the eye was first noted by Schirmer[1] and since his report approximately 20 cases have been reported in the literature. This case is reported because the disciform type of keratitis recurred on two occasions and cortisone appeared beneficial in treatment of the condition.

Case report

The patient was a 37-year-old white housewife with a history of myopia. Ten days before examination she had removed some exudate from the site of a primary cowpox vaccination on her infant's forearm. She could not remember having been vaccinated herself. When seen, the patient complained of intense itching of the right eyelids and discomfort at the angle of the jaw.

Figure 1-a depicts the appearance at the initial visit four days later. Vision was 20/30 with correction. Examination showed marked edema of both lids with vesicles and crusts on the surface and along the lid margins, together with marked conjunctival chemosis and injection and a yellowish discharge in the fornix. The slitlamp revealed slight bedewing of the corneal epithelium. The fundus was unremarkable. Tender preauricular nodes were palpated on the right side. Cultures were sterile. A diagnosis of primary ocular vaccinia was made on the basis of the history and clinical appearance. Prophylactic sulfacetamide ointment was administered two times daily.

Figure 1-b shows the findings 10 days later. The lid and conjunctival lesions had resolved leaving only slight scarring of the upper lid margin. The patient complained of diminution of vision which measured 20/200. Epithelial bedewing was noted, with fairly dense wispy corneal opacities and folds in Descemet's membrane, as shown. The anterior chamber was poorly seen; no large keratic precipitates were present. The pupils were equal and active. Treatment with one-percent atropine and 0.5-percent hydrocortisone was instituted. Dramatic improvement occurred within five days and treatment was discontinued.

Five weeks later, the patient's vision was corrected to 20/20. Punctate opacities could be seen in the area depicted on either side of Bowman's membrane (fig. 1-c), but there was no corneal staining and the patient was asymptomatic.

One month later and 10 weeks after the onset, the patient noted a recurrence of blurring and photophobia. Vision was 20/50—. Incipient corneal vascularization was seen above with a dense disc-shaped corneal opacity and wrinkles in Descemet's membrane (fig. 1-d). The right pupil was smaller than the left. Treatment with cortisone and atropine was instituted.

Ten days later the process was apparently arrested with residual corneal nebulae noted (fig. 1-e).

An interval examination after all treatment was discontinued showed the patient again asymptomatic with normal visual acuity. Slitlamp examination revealed punctate opacities in the area shown (fig. 1-f) adjacent to Bowman's membrane, with no staining and vascularization.

After 21 months without difficulty the patient noted a second recurrence of blurred vision and photophobia. Vision was 20/200. Examination revealed a superficial epithelial defect overlying an area of deeper opacity and a few folds in Descemet's membrane (fig. 1-g). Treatment was instituted with 2.5-percent hydrocortisone alone, with dramatic improvement within a few days. The patient has remained asymptomatic for the past year and present findings are the same as those shown in Figure 1-f. Vision is normal.

Comment

Corneal involvement in ocular vaccinia may be primary in the form of the rare corneal vesicle, or secondary, as in this case. The secondary involvement may take the form of a superficial ulcer, a diffuse deep keratitis, or a localized deep keratitis of the disciform variety. Corneal involvement occurs in approximately one third of the cases of primary ocular vaccinia.[3, 6]

The findings in this patient conform to the criteria for the diagnosis of disciform keratitis, based on Fuchs' text and elaborated upon by Perera, as a complication of ocular vaccinia.[4] Most cases occur as an accident associated with vaccination against smallpox and more frequently in children, as that of Sinaiko,[2] or young adults.

* From the service of Dr. Virgil Casten, Massachusetts Eye and Ear Infirmary, Boston.

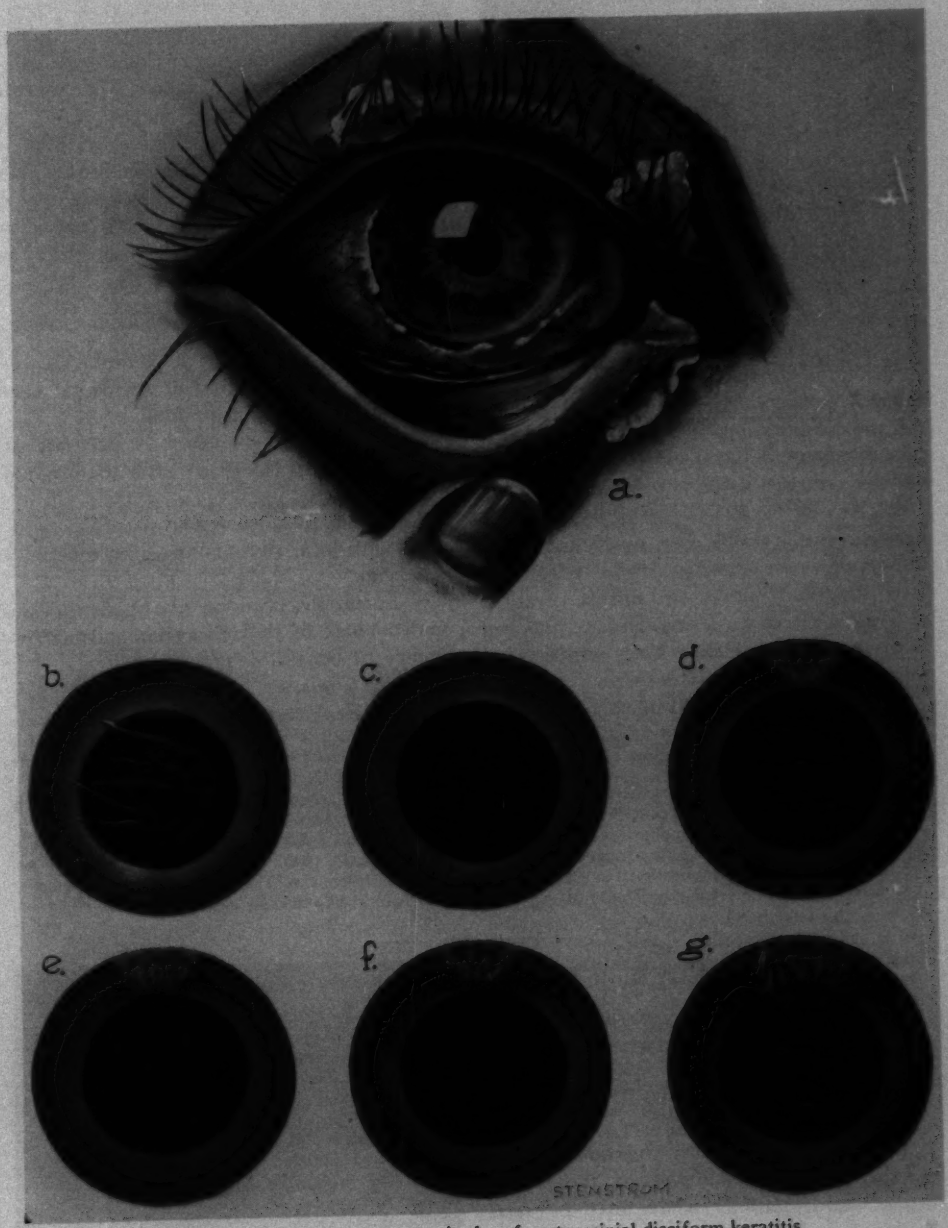


Fig. 1 (Sudarsky). Recurrent episodes of postvaccinial disciform keratitis.

The use of steroid hormones in the treatment of postinfectious viral corneal diseases raises much discussion among ophthalmologists. Many feel that steroids are contraindicated even in late corneal sequelae of virus infections. When other methods have failed, however, steroid hormones have been used on an empirical basis with good results. In this instance the use of cortisone and hydrocortisone produced a rapid improvement in corneal transparency and diminution of signs of inflammation. The administration of cortisone was usually confined to five-day periods.

Sédan[6] has made by far the most valuable contribution to the knowledge of complications of ocular vaccinia. He described in detail the complications arising from the mass inoculation of 800,000 individuals during a smallpox threat in the city of Marseilles in 1952. Among the 19 cases of ocular vaccinia, corneal complications were noted in six. Of these, one was a primary corneal vesicle, two were superficial ulcers, two diffuse keratitis, and one a disciform lesion. Sédan employed cortisone in five of these cases with clinical improvement in four of the five.

In 1947 over 5,000,000 inhabitants of the city of New York were vaccinated. Greenberg[5] reviews the complications, including two deaths, but does not mention ocular disease. Personal communications from several busy New York ophthalmologists failed to reveal any impression of increase in incidence of ocular vaccinia at that time. This dearth of reports is more remarkable in view of the estimate of 74 percent susceptibility of the population of New York prior to vaccination.

Summary

1. A disciform type of keratitis is reported as a complication of accidental vaccination of the lids of an adult.

2. The keratitis recurred at intervals of two months and two years after the original disease process.

3. In each instance a short period of treatment with local steroids appeared markedly beneficial.

4. Some observations are made on the epidemiology of ocular vaccinia and the frequency of corneal complications.

Retina Clinic,
210 East 64th Street (21).

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CAROTID-CAVERNOUS FISTULA*

Report of two cases with their ocular manifestations

John H. Sheets, M.D.
Columbus, Ohio

Two cases of carotid-cavernous fistula are presented as examples of the varied ocular manifestations which may be seen in this disease.

* From the Department of Ophthalmology, The Ohio State University School of Medicine.

Case reports

Case 1

B. W., a 37-year-old white woman, denied eye difficulty prior to a hysterectomy done for fibromyomas. She awakened from the anesthesia with a "vague, peculiar headache." Within a week she noted decreased visual acuity, O.D., edema of the conjunctiva and lids, and proptosis. The venous engorgement gradually subsided, but vision continued to decline. When the left eye started to develop similar symptoms, six weeks postoperatively, she consulted W. H. H.†

Examination revealed a healthy 37-year-old woman except for blood pressure of 160/90 mm.

† William H. Havener, M.D.



Fig. 1 (Sheets). Dilatation of scleral and conjunctival vessels.

Hg and many dental caries. Her right eye (fig. 1) showed considerable dilatation of the scleral and conjunctival vessels but no proptosis. The fundus appearance was consistent with a neuroretinitis, showing edema and hemorrhages about the disc. Along the course of all the veins, except nasally, where the veins were occluded, was an unusual small clumping of hemorrhages (fig. 2). Temporally, there was an appearance simulating old choroiditis in several small areas. She denied light perception in this eye. The left eye had slight conjunctival and lid edema with some superficial and deep injection of conjunctival and scleral vessels. The fundus appeared normal. Visual acuity in this eye was 20/20 on first examination but decreased rapidly and was less than 20/200 at the end of a week. Perimetry of the left eye was normal.

Laboratory reports, medical, and ear, nose, and throat consultations were negative.

The tentative diagnosis of diffuse inflammatory disease (such as uveitis or neuroretinitis) was made, heavy dosages of systemic steroids were prescribed, and within two days there was considerable decrease of her retinopathy and hemorrhage.



Fig. 2 (Sheets). Papilledema with hemorrhages about disc and along veins.



Fig. 3 (Sheets). Arteriovenous fistula outlined between carotid and Cavernous sinus.

At this point the patient described her "headache" more accurately as a swishing noise, and what had erroneously been diagnosed at various times as a pseudotumor and later as papillitis and retinitis was obviously an arteriovenous fistula. A bruit was readily heard, disappearing with pressure over the common carotid. Cerebral angiograms showed a fistula between the internal carotid and the cavernous sinus (fig. 3). A Silverstone clamp was gradually tightened on the common carotid. One week later the fundus of the right (blind) eye showed total occlusion of the central retinal artery and vein, with secondary optic atrophy and retinal pigment degeneration. The left eye returned to normal with 20/20 acuity, and has so remained during a three-month follow-up. There is a right divergent strabismus.

CASE 2

Two weeks following a posterior myocardial infarction, M. T., a 47-year-old diabetic Negress, was seen for a superficial foreign-body type pain in her right eye and right frontoparietal headache. Examination then revealed visual acuity to be: O.D. 20/100; O.S., 20/40. Several microaneurysms and several large exudates were noted in the macular area of both eyes. There was slight superficial punctate staining near the lower limbus, O.D. Tension, O.D., was 25 mm. Hg, and O.S., 23 mm. Hg (Schiøtz). Because of a borderline Schirmer's test (six mm. in five minutes), methylcellulose therapy was instituted.

Two weeks later the staining had disappeared but symptoms remained. At this time she had right ptosis and fixed mydriasis. In addition, there was early rubeosis near the angle, O.D. Tension, O.D., was 23 mm. Hg, and O.S. 14 mm. Hg. Visual acuity remained the same.

The patient did not return for one month and, when seen at this time, the tension was found elevated, O.D., to 39 mm. Hg. There was constriction

of the field in this eye to 15 degrees with a 3/1,000 white test object.

Ten weeks following the initial eye complaints, ptosis was less, mydriasis persisted, rubeosis was increasing, and her headache was now described as a "swishing sensation." Auscultation brought out a bruit over the right orbit. (It is of interest that a pressure difference was noted when taken in the sitting [34 mm. Hg] and reclining [43 mm. Hg] positions.) Pilocarpine had not reduced this tension nor had it affected the mydriasis, O.D. The neurosurgeons concurred in the diagnosis of carotid-cavernous fistula.

The sequence in this case could be postulated as a carotid-cavernous fistula developing in a diabetic with known vascular disease. The proximity of the third nerve to the cavernous sinus accounts for the partial paresis of this nerve. The combination of mydriasis, rubeosis, and venous back pressure led to glaucoma.

University Hospital (10).

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TREATMENT OF EYE PROSTHESIS COMPLICATIONS*

Fenn T. Ralph, M.D.

Darien, Connecticut

Introduction

Use of a prothesis in the eye often results in the production of weeping mucus and an infected socket. The treatment of this condition poses a vexing problem to the ophthalmologist, as well as being a source of great discomfort and cosmetic distress to the patient.

Stimulated by this problem, it occurred to me that a mucolytic agent combined with a potent and well-tolerated antiseptic might alleviate this distressing ocular problem.

Having done a small amount of clinical research with a new mucolytic detergent (superinone), I prepared a combination of superinone and Zephiran (S.5540†).

* This study was aided by the Research Department of the New York Eye and Ear Infirmary, with special credit to the following resident physicians: Orr J. Elder, Thomas Edwards, Robert Weaver, and John Carroll.

† S.5540 was supplied by Medical Research Department, Winthrop Laboratories, New York.

Superinone-Zephiran solution (S.5540)

The solution of S.5540 has the following composition:

Superinone	0.25%
Zephiran chloride	1:5,000
Methylcellulose	0.85%

Superinone is a nontoxic mucolytic detergent that has enjoyed widespread use in preparations used to relieve airway obstruction due to blockage by viscid mucus or mucopurulent exudate.[1] Zephiran, a highly effective antiseptic and mild detergent is bacteriocidal against many common conjunctival pathogens.[2] Methylcellulose gives the solution body and increases its viscosity and adhesive power.

Method of study

The 30 patients included in this clinical investigation had either recently lost an eye or had been wearing a prosthesis for some time. Each patient was supplied with a 30-cc. bottle of the superinone-Zephiran solution (S.5540) and instructed to place one drop in the eye socket: (1) Before putting in the prosthesis, (2) at noon, (3) after removing the prosthesis at night. Each patient reported monthly for three months, and a subjective

CHART 1

OBJECTIVE EXAMINATION OBSERVATIONS

Case No.	Matted Lashes		Red Frothy Lid Margins		Conjunctivitis	
	Before	After	Before	After	Before	After
1	4	0	4	1	3	0
2	4	1	3	0	2	0
3	2	0	1	0	0	0
4	1	0	0	0	0	0
5	4	1	3	0	3	1
6	2	2	2	0	2	0
7	3	2	3	3	2	2
8	3	0	3	0	3	0
9	2	0	2	0	4	1
10	2	2	3	3	2	2
11	3	1	2	0	3	1
12	4	0	3	1	2	0
13	1	0	1	0	0	0
14	3	3	2	2	3	2
15	2	2	2	2	4	2
16	4	2	3	1	3	0
17	1	0	1	0	2	0
18	2	2	2	2	1	1
19	3	1	4	0	4	1
20	1	0	1	0	0	0
21	3	1	2	0	2	0
22	4	2	4	1	4	0
23	2	0	2	0	1	0
24	3	0	2	0	2	0
25	3	3	2	2	2	2
26	4	1	2	0	2	0
27	1	0	1	0	1	0
28	2	2	1	1	0	0
29	4	2	4	0	4	0
30	2	1	1	0	0	0

Legend: 0 = Normal 1 = Mild 2 = Moderate 3 = Moderately severe 4 = Severe Before = Before treatment After = After treatment.

and objective examination was recorded at each visit.

For objective examination observations see Chart 1.

SUBJECTIVE EXAMINATION

1. In 23 of the patients, considerable subjective improvement was claimed from the cosmetic point of view.

2. Five patients said they felt some improvement, and wanted to continue using the superinone-Zephiran solution.

3. Two patients were unable to see any noticeable change in their condition.

COMMENT

This was, of course, a clinical investigation, and subject therefore to all the vagaries germane to such study. However, in view of the fact that, so far as I know at this writing, no other solution has the slightest effect in this ocular malady, it seems worthy of further study, in the hope that a superinone-Zephiran solution may prove a valuable adjuvant in relieving this condition.

SUMMARY

A solution of superinone (0.25 percent) with Zephiran (1: 5,000) and methylcellulose (0.85 percent, S.5540), was used in the treatment of 30 patients wearing prostheses and who suffered from weeping mucus and infected eye sockets.

Subjective improvement in physical comfort and cosmetic appearance was recorded in 25 of 30 patients in a three months' follow-up. Objective observations in the same period showed a decrease in the incidence and degree of matted lashes, of red frothy lid margins, and conjunctivitis.

551 Post Road.

REFERENCES

1. Miller, J. B., and others: Alevaire inhalation for eliminating secretions in asthma, sinusitis, bronchitis and bronchiectasis of adults. Ann. Allergy, **12**:611-626 (Sept.-Oct.) 1954.
2. King, J. H., Jr.: Bacterial infections with reference to traumatic eye injuries: A brief review. Tr. Am. Acad. Ophth., **58**:943 (Nov.-Dec.) 1954.

SIMPLIFIED OFFICE BACTERIOLOGY*

FOR THE OPHTHALMOLOGIST

TED SUIE, PH.D., STANLEY A. SROUFE, M.SC., FRANK W. TAYLOR, M.D., AND W. H. HAVENER, M.D.
Columbus, Ohio

The following report is intended primarily for those ophthalmologists who do not have access to a bacteriologic laboratory and who desire to conduct bacteriologic studies in their own offices. During the past five years we have attempted to find a commercially prepared medium which is suitable for culturing the common ocular bacteria and which, at the same time, is suitable for use in office practice. We have concluded that the culture medium of choice for these purposes is thioglycollate.† We base this on the following reasons:

1. Thioglycollate is a liquid medium; therefore, scrapings and cotton swab smears may be inoculated directly into it. This prevents the desiccation of the clinical material which may result in the destruction of the bacteria.
2. Thioglycollate will suport both aerobic and anaerobic growth.
3. Thioglycollate does not require refrigeration and, in fact, should be kept at room temperature. Prepared tubes may be stored for at least a month without deterioration of the medium.
4. According to our studies, the majority of the pathogens will grow in this medium at room temperature.
5. Thioglycollate is easily prepared by the addition of water to the commercially dehydrated medium.
6. Many of the organisms which are not demonstrable in smears or slides may be cultured in thioglycollate.

The suggested method for bacteriologic studies in the office is as follows:

Scrapings or smears are obtained from the patient suspected of having an ocular bacterial infection. The material is then inoculated into a tube of thioglycollate. The culture tube is allowed to incubate at room temperature or, preferably, if an incubator is available, at 37°C. It is important that cultures are obtained before sulfa or antibiotic therapy is instituted in order to avoid suppression of the growth of organisms.

If bacteria are present, growth will usually occur within 24 hours at 37°C. and within 48 hours at room temperature. The main advantage of taking a culture in this manner lies in the fact that a culture of the organism is available for identification should the patient not respond to the therapy prescribed on the first visit. In vitro antibiotic sensitivity tests may also be performed.[1] It is desirable to obtain smears on slides at the same time as the cultures are made since valuable information may be gained from Gram-stained and Wright-stained smears.

In order to determine whether pathogens will grow in thioglycollate at room temperature, the following investigation was conducted: A total of 126 paired cultures was obtained from the lower cul-de-sac of individuals having primary or secondary conjunctivitis. One tube of the inoculated thioglycollate was incubated at 37°C. and the other was incubated at room temperature.

* From the Department of Ophthalmology, The Ohio State University School of Medicine.

† May be obtained from Difco Laboratories, Detroit, Michigan, or Baltimore Biological Laboratories, Baltimore, Maryland.

TABLE 1

ORGANISMS WHICH FAILED TO GROW AT ROOM TEMPERATURE BUT DID GROW AT 37°C. (15 CULTURES)

Organism	Number of Strains
Streptococcus viridans	6
Staphylococcus pyogenes var. albus (coagulase-negative)	6
Staphylococcus pyogenes var. aureus (coagulase-positive)	1
Staphylococcus pyogenes var. aureus (coagulase-negative)	1
Aerobacter aerogenes	1
	15 from a total of 126 cultures (14.3%)

The following organisms were isolated from thioglycollate incubated at room temperature: Aerobacter aerogenes, Corynebacterium sp. (diphtheroids), Diplococcus pneumoniae, Klebsiella pneumoniae, Proteus vulgaris, Pseudomonas aeruginosa, Staphylococcus pyogenes var. aureus (hemolytic; coagulase-positive), Staphylococcus pyogenes var. albus (hemolytic; coagulase-positive), Staphylococcus pyogenes var. albus (hemolytic; coagulase-negative), Staphylococcus pyogenes var. albus (nonhemolytic; coagulase negative), and Streptococcus viridans.

As indicated in Table 1 a total of 14.3 percent of the cultures which showed growth at 37°C. did not grow at room temperature. A summary of the study is presented in Table 2.

TABLE 2

SUMMARY OF RESULTS OF STUDY

Total number of cultures	126
*Number of positive cultures giving identical results at 37°C. and room temperature	82
†Number of negative cultures giving identical results at 37°C. and room temperature	26
Total number of cultures giving identical results	108
Percentage of cultures giving identical results	85.7

* Positive culture = One which showed turbidity in thioglycollate (identification made by biochemical tests).

† Negative culture = One which showed no turbidity in thioglycollate.

SUMMARY

The clinical diagnosis of bacterial infections is occasionally uncertain. This is especially true if the response to antibiotic therapy is delayed. Under these circumstances a simple culture technique adaptable to office use is helpful. Our results indicate that thioglycollate, a stable culture medium, will support growth of the majority of pathogens at room temperature, and is therefore well suited for office bacteriologic studies.

University Hospital.

REFERENCE

1. Suie, T., Havener, W. H., and Sroufe, S. A.: Brit. J. Ophth., 40:100, 1956.

NEW METHOD FOR ATTACHING EYE SHIELDS*

D. M. SMART, M.D.
Chicago, Illinois

J. W., a 57-year-old Negress, was admitted to the Illinois Eye and Ear Infirmary on February 1, 1955, from the state mental hospital with a diagnosis of bilateral senile cataracts and an unclassified psychosis. The vision in the right eye was hand movements at one foot and the left eye was hand movements at two feet.

On February 3, 1955, an intracapsular lens extraction of the left eye with a full iridectomy was performed. The postoperative course was characterized by unco-operativeness and repeated removal of the protective

* From the Department of Ophthalmology of the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine.



Fig. 1 (Smart). Heavy black-silk skin sutures are placed perpendicular to the border of the shield while it is held in place to act as a guide. In this picture the shield was outlined with methylene blue on the skin.

Fox shield with traumatism to the eye. This occurred despite liberal use of Scotch tape, adhesive tape, elastoplast, heavy sedation, arm restraints, and an ace bandage around the head. The eye developed separation of the wound and iris incarceration. It was enucleated April 21, 1955, because of uncontrollable pain in an irritable, essentially blind eye.

On August 3, 1956, the patient was readmitted for right cataract surgery. Vision was light perception and projection. On August 9, 1956, she had an intracapsular lens extraction with a full iridectomy. Three preplaced corneoscleral sutures of 6-0 mild chromic catgut and interrupted conjunctival sutures were used for the closure. Subconjunctival penicillin and streptomycin were injected.

The eye shield was sewed to the skin with four 1-0 black silk sutures (see illustrations of another patient). The sutures were placed so as to provide maximum stability of the shield. Full skin-thickness bites one quarter of an inch long at the border of the shield and perpendicular to it were taken. The inner suture arm was threaded through a hole in the shield, and the outer arm was brought over the border. The eye was closed and an eyepad placed over the eye. The shield was tied in place and Scotch tape was placed over the shield thus sealing the free ends of the sutures.

The eye was first dressed on the third day—at which time the eyepad was discontinued. Atropine and Neosporin ointment was placed under the shield every day. The shield was removed every third day for examination purposes.

This proved to be an effective method of protecting the eye, as the patient was ob-

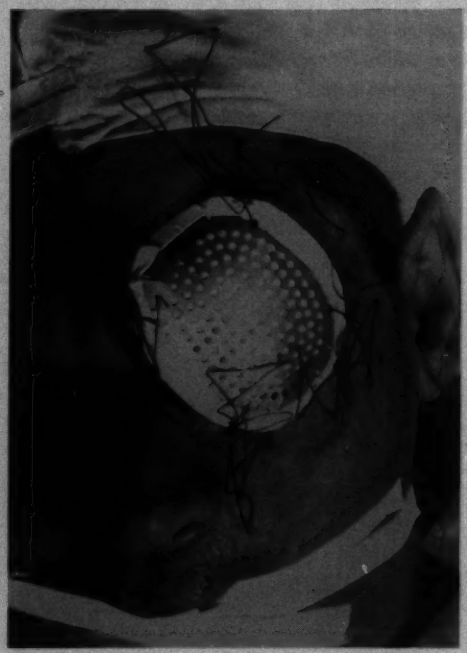


Fig. 2 (Smart). The inner arms of the sutures are brought through a hole in the shield and tied like a shoestring.



Fig. 3 (Smart). The free ends of the sutures are placed centrally on the shield and are covered by Scotch tape. This prevents the patient from untying them.

served to make repeated attempts to rub her eye and manipulate the shield. The slightest tug on the sutures would cause her to remove her hand. The postoperative period was smooth and without complications.

Forty days after surgery the shield was removed permanently. This method has been used on two other psychotic patients with similar results.

It is suggested that anyone using this method make full skin-thickness bites of at least one quarter of an inch in length. The suture should be heavy black silk (1-0) with free ends about five inches long to facilitate tying.

904 West Adams Street.

CYSTICERCUS IN VITREOUS CAVITY*

ALFONSO GAITÁN-NIETO, M.D.

AND

ALFONSO TRIBIN-PIEDIAHITA, M.D.

Bogotá, Colombia

S. de B., a 36-year-old married woman, was first seen in consultation at the hospital in October, 1956, complaining of diminished visual acuity in the right eye for the past six months.

External examination, O.U., was negative. Tension was: O.U., 20 mm. Hg (Schiøtz). Vision was: O.D., fingers at three feet; O.S., 20/20.

Ophthalmoscopy. O.D. showed a globular semitransparent mass whose smooth surface gave emerald greenish reflexes. The mass was well demarcated and surrounded by a reddish hue. It appeared to be mobile at about five disc diameters and was located in the posterior third of the vitreous in front of the optic disc and macula. Occasionally the movements of the parasite had a tendency to pull the mass toward the upper temporal quadrant. Within the mass and near the lower pole, a dense white yellowish formation could be detected. When looking at the formation from a special angle, a sort of protrusion could be seen whose shape changed. It was found to be the scolex. Hanging from the inferior pole of the mass and floating in the vitreous was a small tail.

Laboratory findings. Skin test and complement fixation tests were positive. There was a slight increase of eosinophils. Stool examination was negative, although the patient stated she had observed fragments of possible Taenia solium especially after taking laxatives.

Diagnosis. Ocular cysticercosis.

* From the Ophthalmological Service, Hospital San Juan de Dios, Universidad Nacional de Colombia.

Treatment. The cyst was removed surgically by the scleral approach at about the equator below the lateral rectus. After incision of the conjunctiva, the lateral rectus muscle was severed. An incision was traced in the sclera parallel to the limbus and 12 mm. from it. Prior to the incision, the area was treated with surface diathermy. Then incision of choroid and retina was performed but no spontaneous extrusion of the cyst was noted, as had previously been reported.

Under direct ophthalmoscopy control, the cyst capsule was grasped in the area of the scolex with Arruga's capsular forceps and an intracapsular extraction of the cyst was performed. The amount of vitreous loss was minimal. The scleral incision was closed and an inward scleral buckling was done. Air was injected into the vitreous, the lateral rectus was reattached, and, finally, the conjunctiva was sutured. Penicillin (200,000 u.) was injected subconjunctivally.

Antibiotics, corticosteroids, and mydriatics were prescribed. Pathologic examination of the cyst proved it a typical cysticercus of Taenia solium. Postoperative reaction was minimal. About the 10th or 12th day, when the media cleared, the fundus could be examined. The retina appeared detached. A scleral buckling was performed with an uneventful recovery.

Six months later, vision was: O.D., 20/400. The fundus showed the scars from the diathermy, the scleral buckling, and the large macular scar where the cyst was implanted.

Calle 22, No. 4-75.

Ophthalmic Miniature

The advantages of the corneal flap extraction may be much enhanced, and its dangers materially lessened, in my judgment, by the use of a suture to retain in apposition the edges of the wound. Securing a more immediate union, we not only avoid ulceration of the border of the flap, and prolapsus iridis with it attendant evils, but the prompt restoration of the fullness of the globe, and of the normal relations of its several parts, lessen the chances of irritation, from pressure of any cortical fragments or remnants of capsule upon the delicate continguous structures, and the occurrence of iridocyclitis. This suture, a simple strand only of the finest glover's silk, passed through the edges of the wound by means of a very minute, short needle, held by forceps, can be tolerated, without detriment, even in the cornea.

"Cataract extraction operations," by Henry W. Williams, *Archives Ophthalmology and Otology*, 1:102, 1869.

OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

FIGHT FOR SIGHT AWARDS
of the
NATIONAL COUNCIL TO COMBAT BLINDNESS, INC.

Grants-in-aid, predoctoral and postdoctoral research fellowships, travelling fellowships, summer fellowships (primarily to students in medicine and the basic sciences) as approved by the Scientific Advisory Committee, June, 1957

Grants-in-Aid

INVESTIGATOR	INSTITUTION	PROJECT TITLE	AMOUNT
Adler, Francis H., M.D. and Frayer, William C., M.D. (continuation)	Department of Ophthalmology, Hospital of the University of Pennsylvania	"Study of the factors involved in the proliferation of the retinal pigment epithelium in disease"	$3,500
Balazs, Endre A., M.D. (continuation)	Retina Foundation, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary, Harvard Medical School	"Studies of the hyaluronic acid formation in the vitreous body"	3,700
Ballen, Peter H., M.D.	Department of Surgery, Division of Ophthalmology, State University of New York, College of Medicine at New York City, Brooklyn	"Mucous membrane grafts in lye (chemical) burns with reference to method of healing action and vascularization of cornea"	2,000
Bettman, Jerome W., M.D. and Fellows, Victor, M.D.	Department of Surgery, Division of Ophthalmology, Stanford University Medical School, San Francisco	"The effect of pressure, including tonography, on the intraocular blood volume"	2,750
Breinin, Goodwin M., M.D. (continuation)	Department of Ophthalmology, New York University Post-Graduate Medical School, New York University-Bellevue Medical Center	"Electromyography of the extraocular muscles, including stimulation studies and evaluation of drug effects on the action current"	2,500
Burns, Robert P., M.D.	Department of Ohpthalmology, College of Physicians and Surgeons, Columbia University, New York	"Tissue culture of generalized cytomegalic inclusion disease virus with electron microscope studies"	1,000
Byerly, Baxter H., M.D.	North Carolina Memorial Hospital, University of North Carolina School of Medicine	"A study of bizarre corneal disease indigenous to American Indians in eastern North Carolina"	550
Cibis, Paul A., M.D. (continuation)	Department of Ophthalmology, Washington University School of Medicine, St. Louis	"Histopathology of the eye with oblique illumination"	5,600
Drance, Stephen M., M.D.	Purchase of equipment for use by investigator now in Medicine Hat, Alberta, Canada, for study in collaboration with Dr. Antoinette Pirie, Nuffield Laboratory, University of Oxford, England. Project	"Study of lens epithelium following irradiation"	1,000
Eliasson, Sven G., M.D.	Department of Internal Medicine, The University of Texas Southwestern Medical School	"The role of visual impulses in the control of eye muscle activity"	5,000
Ellerbrock, V. J., Ph.D. (continuation)	School of Optometry, Ohio State University	"Preparation of volume on aids for persons with subnormal vision"	500
Garron, Levon K., M.D. and Foerster, Helen Campbell	Francis I. Proctor Foundation for Research in Ophthalmology, University of California Medical School	"Special histopathologic study of endogenous uveitis"	4,400
Gordon, Dan M., M.D. (continuation)	Department of Ophthalmology, New York Hospital, Cornell Medical Center	"Application of visual aids in patients with subnormal vision"	3,675
Green, Harry, Ph.D.	Research Department, Wills Eye Hospital, Philadelphia	"Osmotic pressure of intraocular fluids"	3,500
Green, Harry, Ph.D. (continuation)	Research Department, Wills Eye Hospital, Philadelphia	"Intraocular pressure and the bicarbonate ion concentration in animal eyes"	5,000
Hepner, Ray, M.D.	Department of Pediatrics, University of Missouri School of Medicine	"The effects of maternal hyperoxemia on the intrauterine development of the eye"	1,000
Jacobson, Jerry H., M.D. (continuation)	Division of Electrophysiology, New York Eye and Ear Infirmary	"Electrophysiology of the eye"	2,500

Investigator	Institution	Project Title	Amount
I. C. Michaelson, M.D. and Blumenkrantz, Nelly, Ph.D. (continuation), Department of Ophthalmology, Hadassah University Hospital, Jerusalem, Israel: "(a) Mucopolysaccharide content of vitreous humor in normal and abnormal conditions (b) Mucopolysaccharide content of the conjunctival secretion in certain diseases of the conjunctiva"			3,600
Mitsui, Yukihiko, M.D. (continuation), Department of Ophthalmology, Kumamoto University Medical School, Kumamoto, Japan: "Study of trachoma virus by cultivation and electron microscopy"			1,200
Pogell, Burton M., Ph.D. (continuation), Wilmer Ophthalmological Institute, Johns Hopkins University Medical School, Baltimore: "Metabolism and turnover of mucopolysaccharides in cornea"			4,000
Schueler, Fred Warren, Ph.D., Department of Pharmacology, Tulane University School of Medicine, New Orleans: "The effect of hemicholiniums on the cholinergic functions of the eye"			1,100
Smelser, George K., Ph.D. (continuation), Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York: "The relation of the exophthalmogenic and thyrotropic pituitary principles"			1,800
Tsutsui, Jun, M.D. (continuation), Department of Ophthalmology, Okayama Rosai Hospital, Okayama University, Okayama, Japan: "Clinical and virologic studies in trachoma"			1,800
Wolken, Jerome J., Ph.D. (continuation), Biophysical Research Laboratory Eye and Ear Hospital, University of Pittsburgh Medical School: "Photoreceptor structures"			2,700

Predoctoral and Postdoctoral Research Fellowships

Investigator	Direct Supervisor*	Institution	Project Title	Amount
Barishak, Y. Robert, M.D.	(Michael J. Hogan, M.D.),	Francis I. Proctor Foundation for Research in Ophthalmology, University of California:	(a) "Implantation of tumor tissue into the anterior chamber of animals"; (b) "Investigations on the tissue culture of intraocular tumors, particularly malignant melanomas"	$3,600
Bishop, Harding E., B.Sc.	(Austin H. Riesen, Ph.D.),	Department of Psychology, University of Chicago:	"Are innately determined connections between retina and brain responsible for vision?"	2,700
Farkas, T. G., A.B. (continuation)	(Normand L. Hoerr, M.D.),	Department of Anatomy, Western Reserve University, Cleveland:	"Role of insulin in the metabolism of the lens"	3,600
Krishna, Narendra, M.B.B.S.	(Irving H. Leopold, M.D.),	Department of Physiology and Pharmacology, University of Pennsylvania, Graduate School of Medicine:	(a) "Influence of drugs on ocular function"; (b) "Attempts to produce diabetic retinopathy in experimental animals"	3,300
Maurice, David M., Ph.D.	(Michael J. Hogan, M.D.),	Francis I. Proctor Foundation for Research in Ophthalmology, University of California:	"Transfer of proteins between blood and intraocular fluids"	5,000
Modrell, Robert W., B.A., M.A.	(Albert M. Potts, M.D.),	Department of Surgery, Western Reserve University, Cleveland:	"Studies on electrophysiology of the eye"	2,500
Norton, Herman J., Jr., M.D.	(Irving H. Leopold, M.D.),	Graduate School of Medicine, University of Pennsylvania:	"Participated in advanced training program for certified ophthalmologists, in research, teaching and administration techniques"	1,200
Pratt-Johnson, John A., M.D. (continuation)	(J. W. Bettman, M.D.,	Department of Ophthalmology, Stanford University Medical School, San Francisco:	"Corneal transplantation with particular reference to elimination of delayed sensitivity reaction as a result of homotransplantation: Metabolism and turnover of polysaccharides in cornea and lens, as well as other chemical and allergic reactions which take place in the recipient's eye"	4,800

Traveling Fellowships

Investigator	Institution	Purpose	Amount
Haig, Charles, Ph.D.,	Department of Physiology, New York Medical College:	Under a National Council to Combat Blindness Grant-in-Aid, this investigator conducted a study entitled "Certain correlates of structural to functional parameters for the luminence threshold of the human eye." This Traveling Fellowship enables him to accept a special invitation from Dr. G. B. B. M. Sutherland to attend a symposium on visual problems of color. Attendance at this symposium, where colleagues from nine countries will discuss problems in this scien-	

* All Direct Supervisors' names are enclosed in parentheses.

tific area, will be of great value to the investigator in the preparation of papers for publication $520

Wolken, Jerome J., Ph.D., Biophysical Research Laboratory, Eye and Ear Hospital, University of Pittsburgh Medical School: A grantee of the National Council to Combat Blindness for the past four years, this fellowship is enabling him to visit institutions in Italy, Denmark, Sweden, and the British Isles, to consult on the problem on which he is engaged with other experts in the field. Project: Photoreceptor structures 1,500

Summer Fellowships

(This facet of the research program of the National Council to Combat Blindness was designed to promote interest in ophthalmic research among students in medicine and the basic sciences)

Investigator	Direct Supervisor*	Institution	Project Title	Amount

Alexander, Richard K. (L. Vernon Scott, Sc.D.), Department of Microbiology, University of Oklahoma, School of Medicine: "Latent herpes simplex infection of the eye" $600

Applebaum, Seymour (Max Chamlin, M.D.), Department of Ophthalmology, Albert Einstein College of Medicine: "Problems of electrophysiology and retinal detachment" 400

Bard, Leslie A. (Burton H. Pogell, M.D.), Wilmer Ophthalmological Institute, Johns Hopkins University, Baltimore: "The role of mucopolysaccharide metabolism in the pathogenesis of diabetic retinopathy" 600

Benoit, Richard P. (Alfred Marshak, Ph.D.; Wm. Stone, Jr., M.D.), Massachusetts Eye and Ear Infirmary, Marine Biological Laboratory: "Radioautographic studies on the problem of chromosome replication in the endothelium of marine animals" 600

Berman, Robert W. (Harry A. Feldman, M.D.), Department of Preventive Medicine, State University of New York at Syracuse: "Relationship of Toxoplasma infections to congenital and acquired chorioretinitis" 600

Crawford, Joseph B. (Samuel J. Kimura, M.D.), Francis I. Proctor Foundation for Research in Ophthalmology, University of California School of Medicine: "Studies on experimental herpes simplex uveitis" 600

Dickerson, John W. (Wolfgang A. Lieb, M.D.), Department of Ophthalmology, Medical College of Virginia: "The immune response in corneal homo- and hetero-transplants" 600

Farr, James H. (Milton Flocks, M.D.), Department of Ophthalmology, Stanford University School of Medicine: "The influence of trabeculectomy on the facility of aqueous outflow" 600

Giles, Kenneth M. (John E. Harris, Ph.D.), John E. Weeks Memorial Laboratory, University of Oregon Medical School: "The movement of insulin across blood-aqueous humour 600

Gondelman, Martin (Joseph Mandelbaum, M.D.), Department of Surgery, Division of Ophthalmology, State University of New York, College of Medicine, Brooklyn: "Dark adaptation" 400

Jewett, Robert E. (Harold F. Falls, M.D.), Department of Ophthalmology, University Hospital, University of Michigan Medical School: "The social, economic and hereditary aspects of the blind marrying the blind" 400

Jones, Helen S., Ph.D. (Alfred Marshak, Ph.D.; Wm. Stone, Jr., M.D.), Massachusetts Eye and Ear Infirmary; Marine Biological Laboratories: "Investigation of the composition of the aqueous humour of marine Teleosts and Elasmobranchs in relation to theories of the mode of its formation" 600

Keenan, George J. (Earl Fisher, Ph.D.): Department of Ophthalmology, Tulane University School of Medicine: "A study of the mechanism by which cellular materials produced by staphylococci are able to produce ocular damage" 600

Lieberman, Theodore (R. M. Fasanella, M.D.; Bernard Zuckerman, M.D.), Department of Surgery, Section of Ophthalmology, Yale University School of Medicine: "Corneal reactions to iron in foreign bodies" 500

Moyers, Thomas G. (L. K. Garron, M.D.), Francis I. Proctor Foundation for Research in Ophthalmology, University of California School of Medicine: "Electronic microscopic studies on the trabecular meshwork of the human eye" 500

Oglesby, Richard B. (Bernard Becker, M.D.), Department of Ophthalmology, Washington University School of Medicine: "Uptake of radioactive sulfate by the cornea" 600

Reinecke, Robert D. (David G. Cogan, M.D.), Howe Laboratory of Ophthalmology, Massachusetts Eye and Ear Infirmary, Harvard University Medical School: "Quantitative variations of intraocular pressure due to autonomic innervation" 600

Rubin, Herbert S. (Goodwin M. Breinin, M.D.), Department of Ophthalmology, New York University College of Medicine and New York University Post-Graduate Medical School: "Electrophysiologic studies in ocular motility" 500

* All Direct Supervisors' names are enclosed in parentheses.

Investigator	Direct Supervisor*	Institution	Project Title	Amount
Rubin, Walter	(Edward W. D. Norton, M.D.),	Department of Ophthalmology, Cornell University Medical College:	"Studies related to the gelling capacities of the vitreous	600
Sabesin, Seymour M.	(Ralph Z. Levene, M.D.)	Department of Ophthalmology, New York University Post-Graduate Medical School:	"A study of ocular blood flow in experimental animals"	400
Shamblin, James R., Jr.	(F. W. Schueler, Ph.D.),	Department of Pharmacology, Tulane University School of Medicine, New Orleans:	"The effect of hemicholiniums on the eye"	600
Srebro, Richard	(Bernard Becker, M.D.),	Department of Ophthalmology, Washington University School of Medicine, Saint Louis:	"Improved methods of electronic tonometry"	600
Terry, William D.	(Peter H. Ballen, M.D.),	Department of Surgery, Division of Ophthalmology, State University of New York College of Medicine, Brooklyn:	"Mucous membrane grafts in lye (chemical) burns with reference to methods of healing action and vascularization of cornea"	600
Wolfe, Richard L.	(Ronald Wood, Ph.D.),	Wilmer Ophthalmological Institute, Johns Hopkins Hospital, Baltimore:	"Pathogenesis of ocular diseases: An experimental determination of antibody titers to streptococci in serum from patients with granulomatous uveitis"	600

THE ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY

Future Meetings

EAST CENTRAL SECTION:	Cleveland, Ohio, January 7, 1958 Albert M. Potts, M.D., chairman, University Hospitals, 2065 Adelbert Road, Cleveland 6, Ohio
EASTERN SECTION:	Bethesda, Maryland, January 17, 18, 1958 Ludwig von Sallmann, M.D., chairman, National Institute of Neurological Diseases and Blindness, Bethesda 14, Maryland
MIDWESTERN SECTION:	Saint Louis, Missouri, April 21, 1958 Bernard Becker, M.D., chairman, 640 South Kingshighway Boulevard, Saint Louis, Missouri
NATIONAL MEETING:	San Francisco, California, June, 1958 Lorand V. Johnson, M.D., secretary, 10515 Carnegie Avenue, Cleveland 6, Ohio

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

CHICAGO OPHTHALMOLOGICAL SOCIETY

January 21, 1957

DR. KENNETH ROPER, *president*

EARLY COMPONENTS OF CORNEAL WOUND CLOSURE

DR. JAMES E. MCDONALD presented an analysis of the factors operative in wound healing in the first hour after a perforating corneal wound. In a series of rabbits an incision five mm. long was made in the central portion of the cornea. The events occurring in the first hour were continuously observed with a high-power slitlamp and at varying intervals the eyes were enucleated for histologic study. This procedure was repeated in a second series of rabbits that had been previously heparinized and in a third series that were killed by intravenous air injection just prior to the corneal incision. These experiments were repeated in the cat, dog, monkey, and in five human eyes prior to enucleation.

Dr. McDonald demonstrated by means of photomicrographs and drawings that early wound closure results from fibrin plugging of the wound rather than from corneal edema. This fibrin plug can be prevented by heparinization and indeed a permanent flat anterior chamber can be so induced with constant seepage of aqueous through the wound.

Discussion. DR. LESLIE B. AREY: I have been interested in some aspects of this problem for a good many years. Some 40 years ago I started to work on an investigation of the eye and have continued it somewhat intermittently ever since. Wound healing in some aspects also has held my attention over many years, and a different phase of corneal wound healing, which I may mention since it is bound up with this particular problem too, is also something which I had the pleasure to investigate.

I think that Dr. McDonald has practically eliminated, in a sense, any discussion, except praise, because his investigation has been pursued in an ideal way. He seemingly has wrapped it up quite tightly, and has proceeded both by microscopic and macroscopic means to arrive at very sound conclusions. Let me just comment on a few things that he said, and they will perhaps be more in the way of tying in what he said with fundamental and very well-established principles of wound healing rather than being at all destructive or critical, because I find it very difficult to find vulnerable points in his presentation.

The presence of fibrin and its source as he has presented these data to you, I think is wholly convincing. It seemed to me that the conjunctiva as a possible source of fibrin was ruled out by his correlational observations; that is, the rapid plugging correlated with little fibrin in the conjunctival sac and the leaky plug with much fibrin in the sac, that was rather telling evidence. The aqueous source seemed to me to be equally well supported by the experiments in which he produced a lesion both posteriorly and anteriorly and found the fibrin only where the cornea had been insulated on the posterior aspect.

Now the role of fibrin in closing wounds is one of the soundest principles of wound healing. It occurs, in some form or other, in every type of wound healing, even the slightest wound. It is the sort of thing that you get if you just nick your face with a razor, and then compress the parts into apposition. If you examine these lesions microscopically you will find that there is a plate of fibrin formed. In every surgical apposition of parts there is a plate of fibrin formed. In closure by scab formation in somewhat more intensified wounds, the fibrin, of course, plays a major role.

Now, then, the presence of a fibrin plug, in a gaping wound such as there must be in a perforating wound of the cornea is what you would expect should happen, and what he has demonstrated so nicely, both by slitlamp and microscopic sections, actually did happen. Fibrin is necessary. There must be fibrin in order that the migrating cells, both epithelial and connective tissue cells, have something to crawl on. Tissue-culture experiments, putting glass chambers in the ear, and regenerative and developmental data, all point to the fact that a substrate such as fibrin is a necessary thing for ameboid cells.

In the first hour, to which his experiments are confined, you would expect to find exactly what he did find. There was a complete gaping wound, the only thing that can go in there at that time would be fibrin. The fact that it merely serves as a plug, which he emphasized at this stage, is again what might be expected in the early hours. The primary purpose of such fibrin is glueing or tying things together, whereas the role of fibrin as a highway over which cells can migrate is a much later phase in wound healing. He found little tendency for these edges to approximate and, again, that could have been expected, a priori, the cornea being what it is, with an elastic membrane below, I think you could have predicted a wound of somewhat pyramidal or conical shape such as he found. In any event, you would not expect an approximation of the wound edges in the first hour.

Considering wounds in general, what are the factors that lead to the closure of wounds: (1) is it the migration of cells, both epithelial and connective-tissue cells, (2) or the proliferation of cells, (3) or volume increase of the cells themselves. Dr. McDonald spoke of it as edema; of course, in a cell-poor tissue like cornea, it apparently is edema. But the cells themselves, in wound healing, can swell and, as a matter of fact, there can be an increase up to around 35 percent of volume just by cell imbibition in this region.

Fourth, and last, is contraction. Contraction is a tremendous factor, pulling large wounds together. It is somewhat different in loose skin and in tight skin, but, in all those cases, it is a late phase and it usually has to do with a contraction involving the substrate, or a contraction involving the regression of the cicatrix itself.

Now, if I may just for a moment refer to one or two of those factors: (1) the migration of cells, (2) the proliferation of cells. I became interested quite a number of years ago in the relative roles of migration and proliferation. If you looked in the books published around 1920 or 1930 they still spoke of wound healing, and the actual closing over of the epithelium in particular, as being a process of proliferation. That is, proliferation took place, the new cells filled in, and that is all there was to it. There have been known some things to the contrary: for example, in the healing of the endometrium after the menstrual slough, there is a very quick crawling out of cells from the gland and a resurfacing. You don't find any cell mitosis there; in fact, although they didn't know the reason, Novak and Telinde commented on their inability to find mitosis and were very much disturbed by this. Of course, they found them a little later in what is now known as the estrogen phase, but not during the time of healing. I entered this controversy very curiously because at one time, in the early 1920's, I was doing some investigation for the Bureau of Fisheries. It had to do with the pearl mussel and its larval stage, which often encysts on the gills of the salamander. I was interested in the very quick way in which the little cysts would form. The cysts were not any bigger than a pinhead and the cells, of course, are very large in this animal, but I figured that in housing over each little larva of the mussel there would be something like 320,000 cells formed. In half an hour or so the cyst was pretty well formed; in a few hours it was complete. When you examined those cysts you found practically no mitosis until they were completely made; only then did

they increase greatly to replace the depletions due to the migratory cells. As a matter of fact, in the week following the encystment the rate of mitosis increased some 135 times over what it was in the normal animal.

A number of years later, we became interested in this same phenomenon in respect to the cornea, but these were not perforating wounds. What we wanted to discover was what epithelium would do in corneal healing, and we wanted to make a little controllable defect in the corneal epithelium. We tried all sorts of things and finally hit on the little sand paper discs the dentist uses in the end of his rotating drill. We found that by touching that to the cornea of rats we could produce a well-regulated wound that would be about three mm. long and about 0.3 mm. wide; retraction would increase the width to about 0.5 mm. so we had a wound about three mm. long and 0.5 mm. wide. This area was only about 1.5 sq. mm., but that was about seven percent of the total corneal area. Now, in six hours the denuded floor was half covered; at 12 hours the healing was complete, and after that you had to take special pains to be able to locate in sections where the defect had been made. The real thing we were interested in, as in the salamander, was just what role the mitosis played, because there were various reports as to the mitotic activity in the healing of corneal wounds. We first sought to find about how many mitoses there are in a cubic mm. of normal corneal epithelium; it comes out to just short of 17,000. We were rather struck by these results, we thought 17,000 cu. mm. was large. So to get a comparison we took the esophageal epithelium of a rat, discarded the upper squamous cells and had the same type of epithelium to measure that was encountered in corneal epithelium. The number was something less than 5,000 sq. mm., so mitosis is, in truth, fairly rapid in corneal epithelium. Now you may be interested in the rate of mitosis: the ordinary rate would hold at about 17,000 at the start of the lesion; the number would then drop off a great deal within six hours and at 12 hours it had declined to about 6,000 or 7,000. In 24 hours it came back a little, and at 48 hours it was just a bit higher. Here it maintained a plateau until about the 75th hour. At 96 hours it came up to about 14,000; at 120 hours it rose to 26,000; and at 192 hours it was back to about 17,000, and then in about 10 days it was even less than normal.

The response always in the epithelial healing of a small wound was a quick drop off in mitotic rate, like a shock reaction following the wound to a level far below normal. This low rate was maintained for a period of time after the epithelial healing was complete. Then the rate would begin to rise and finally exceed the normal. Finally it would decline, actually for awhile going below the normal but eventually returning to normal. That is the general epithelial role in small wounds.

Again, in closing, let me say that I think Dr. McDonald is to be highly complimented on the way he has done this investigation and on the nice way he presented it.

Dr. William F. Hughes, Jr.: Unlike Bender and Bender, who also showed the importance of fibrin in rabbits, Dr. McDonald has gone a step further and shown, which I think has not been shown before, that fibrin is important in monkey and human corneal wound healing. I believe the virtues of fibrin have been well pointed out: (1) it seals the anterior chamber almost immediately, (2) it keeps the corneal epithelium from growing down into the anterior chamber, certainly a fine virtue, (3) it provides a scaffolding across which the fibroblast from the surrounding cornea may grow.

Are there any deficiencies in the function of fibrin? Would Dr. McDonald comment on the fact that certain individuals claim that wounds that heal by fibrin plug are weak? Weaker than those which do not?

After extolling the virtues of fibrin is he going to discontinue the use of corneoscleral

sutures in cataract extraction?

Does fibrin in the anterior chamber tend to form more posterior and anterior synechia? If so, perhaps we will have to re-evaluate the use of paracentesis in such conditions as iritis, particularly those with secondary glaucoma. The fibrin might well make the secondary glaucoma worse.

Another unwanted presence of fibrin has been pointed out by Dr. McDonald; after filtering operations. Some years ago Beck and Haines showed that the subconjunctival injection of herparin did not seem to aid in the filtration of glaucoma operations in rabbits.

So, in view of Dr. McDonald's findings, it would seem that systemic heparinization would probably be necessary to inhibit fibrin formation in the anterior chamber and the posterior part of the wound.

One wonders if this could be done safely without the danger of excessive hemorrhage at operation. Possibly so, because hemostasis is thought to depend on other factors more important than the formation of fibrin, which could block hemorrhage. For example, reflex contraction of the vessel wall, liberation of vasoconstrictor and hypotensive substances from the platelets, and the stickiness of the endothelium of the capillary walls which tend to seal it off.

In closing I want to compliment Dr. McDonald very much for carrying out such a nice piece of basic work which has so many practical implications.

DR. MCDONALD (closing): Does fibrin precipitate synechias, such as in a paracentesis?

I expect that it does and I think that if a paracentesis is done, it isn't too uncommon, at least in animals, to get an anterior synechia at the site of the paracentesis, especially if it is done in the periphery.

As Dr. Hughes mentioned, the work of Beck and Haines in promoting the fistulization in glaucoma operations in animals was done with a local instillation of heparin which I do not think would be adequate because the secondary aqueous is constantly being reformed from the blood vessels of the iris, and from other sources.

He brought up the point, wondering if systemic heparinization is safe in humans. I don't know if it is safe, and we are certainly not advising it. We are just using this technique in our program in trying to elucidate certain things about healing. Also, in reference to hemorrhages, I don't think I mentioned this in the paper, but in the heparinized dog, it was very interesting. When you made the incision, at two or three different points of the iris, well away from the site of the incision, there were noted little petechial hemorrhages and these hemorrhages then mixed in the aqueous stream, so that the rapid movement of the aqueous was dramatized. You could see these little hemorrhages come forth, pour out through the wound and down over the cornea. We felt that this was just another step in capillary permeability, the first step being the formation of a protein beam, the second a more dense fibrinous response, and the third the pouring of red blood cells into the aqueous stream.

We didn't feel that it was a hemorrhage caused by direct trauma; rather, it was a hemorrhage caused by decompression. This was noted in one human patient too.

Are wounds that are healed by fibrin weaker than other wounds?

I believe that all wounds are healed by fibrin. Even those that are sutured. I believe that when you suture a wound, you put the stromal edges together and naturally you have a stronger wound.

I think it is much better to have the stroma close together than to have a narrow bridge of fibrin between two, almost but not quite, abutting edges of the stroma. I think you'll have a stronger wound that way than if you have a wound that has a fibrin bridge between them.

No, I am not planning to abandon corneoscleral sutures—and finally when I alleged that fibrin blocked the ingrowth of epithe-

lium, I didn't mean to imply that we concluded that from our work. That was concluded from the work of Bender and Bender. They maintained heparinization in these animals by twice daily injections of heparin and, as a consequence of this, the epithelium proceeded down the cornea, completely covering the wound and started to grow on the back of the cornea.

But our work, of course, would not show that. The only thing in the first hour was just a little invasion of the epithelium.

Ophthalmodynamometry and intracranial disease

Dr. Everet H. Wood (Madison, Wisconsin) presented a series of cases in which measurement of the blood pressure in the central retinal artery was of aid in diagnosing intracranial disease. He stated that only diastolic measurements were used and because of variation in readings from time to time in the same patient absolute readings were required and only differences of 10 mm. Hg or more between the two eyes were considered significant. Ophthalmodynamometry is most useful in confirming a diagnosis of unilateral carotid artery disease.

Discussion. Dr. Robert W. Hollenhorst (Rochester, Minnesota): I am grateful to Dr. Wood for having sent me his interesting paper to review. I am pleased to be asked to comment on his paper, because our group has been much interested in ophthalmodynamometry ever since Dr. Henry Wagener brought back one of Bailliart's instruments from the 1938 International Congress held in Cairo, Egypt. It seems to me that we are much more impressed with the ophthalmodynamometer than Dr. Wood professes to be. I have used this instrument off and on for more than 11 years, but it was not until 1952 that I learned to appreciate its value in diagnosis of vascular disease, and especially that of vascular disease in the carotid system. Dr. Hendrik Svien, one of our neurosurgeons, and I have been particularly interested in measuring the changes in the retinal arteries which follow surgical ligation of the branches of the common carotid artery. We have just published a paper on this subject in the *Proceedings of the Staff Meetings of the Mayo Clinic*. Dr. Millikan, Dr. Siekert, and Dr. Whisnant of our Section of Neurology and Dr. Ruker, Dr. Kearns, and I of the Section of Ophthalmology have found this instrument particularly valuable in the management of patients after strokes.

Before going any further, I shall mention that we use the diastolic readings almost exclusively, as they are consistent in successive tests and thus more reliable than the systolic readings. If the diastolic readings are less than 50 mm. Hg, we consider a five-mm. difference between the two eyes significant; if more than 50 mm., we consider a difference of 10 mm. significant. Systolic pressures are measured only if the diastolic pressures are equal when a carotid lesion is suspected, or if the differences in diastolic level are less than our five-mm. to 10-mm. requirement. If the diastolic levels are near 20 when the patient is sitting, we raise the pressure for greater accuracy by placing him in the supine position. We usually take from three to 10 readings of each eye; diastolic pressures are not affected appreciably by successive readings as systolic pressures are.

In addition to the unilateral lowering of pressure in the retinal artery which is a reflection of lowered pressure in the internal carotid artery, evidence of several other interesting phenomena may be observed with the ophthalmoscope. Dr. Kirby and I, in 1935, described two patients with hypertension in whom unilateral carotid ligation had previously been carried out for bleeding intracranial aneurysm: one of these patients had normal retinal blood vessels on the ligated side, but moderately severe hypertensive changes on the other side; the other patient had mild hypertensive retinal changes on the ligated side, but had papilledema, exudates, and severe vascular changes of hy-

pertension, group IV, on the other side and eventually died of malignant hypertension. We have observed this phenomenon at least a dozen times since the first report. Six of 55 patients with intermittent insufficiency of the internal carotid artery had this manifestation.

A second phenomenon is most interesting and has been observed repeatedly with the aid of the ophthalmoscope in these patients, namely, the appearance of ischemic phenomena in the form of cotton-wool patches, with or without visual impairment, on the side with the lowered pressure in the retinal artery.

Now I should like to discuss briefly our findings among several different groups of patients: (1) those who had unilateral occlusion—spontaneous or surgical—of the internal carotid artery, (2) those who had unilateral ligation of the common carotid artery, (3) those who had intermittent insufficiency of the internal carotid artery.

1. We have studied 11 patients in the early postoperative period following ligation of the internal carotid artery. In each instance a significant difference between the two sides was found.

The pressures in the two retinal arteries at various intervals after ligation of the right or left internal carotid artery were studied in six cases and again a significant difference was found.

Angiograms demonstrated occlusion in the cervical region of one internal carotid artery in four cases studied. In every instance a significant difference was found in the pressure in the two retinal arteries, and I may say that we have had no case in which angiograms demonstrated normal carotid circulation if retinal arterial pressures were unequal.

However, if the internal carotid artery is occluded distal to the orifice of the ophthalmic artery, retinal arterial pressures may be equal on the two sides. We studied one such case. An arteriogram made of the right internal carotid artery in a patient on June 17, 1955, demonstrated occlusion of the internal carotid artery in its siphon portion distal to the origin of the ophthalmic artery. Retinal arterial pressures determined the next day were equal on the two sides, measuring 45 mm. of mercury on each. Nine months later, another angiogram was made which demonstrated occlusion of the right internal carotid artery just beyond the carotid bulb. Determinations of the retinal arterial pressures the next day revealed a significant difference; pressure was 50 mm. of mercury on the right and 35 on the left.

2. We studied six cases in the early postoperative period after one common carotid artery was ligated. In five of these, significant differences in diastolic pressure were present; but in one case the difference was only five mm. of mercury. In some of these cases, the internal carotid artery on the same side was ligated a week later. Pressure decreased further on this side. The presumption is that, if the internal carotid artery is patent, sufficient blood flows through the external carotid artery in a reverse direction into the internal carotid artery to provide about one half of the normal pressure in the ophthalmic and retinal arteries. After the internal carotid artery is ligated, flow from the external carotid artery is not possible except through intraorbital anastomosis, and the pressure is significantly lowered in both the ophthalmic and retinal arteries. Two of these patients had inordinately great differences in pressure after the common carotid artery was ligated. Seven to 10 days after the ligation, neither of them tolerated occlusion of the internal carotid artery. On release of the ligature around the internal carotid, both patients recovered.

We studied four cases four months to three and one-half years after ligation of a common carotid artery. In one case, retinal arterial pressures were obtained within the early postoperative period and again four months later. The difference had decreased from 18 (50 to 32) to 10 (50 to 40) in those four months. Another of these cases showed

no difference between the pressures in the two retinal arteries one and one-half years after ligation of the common carotid artery. In the third case the difference was only 6.0 mm. after three and one-half years; and in the fourth, the difference was 10 mm. one year after ligation (when this patient's internal carotid artery was compressed, the pressure dropped to 32 mm. Hg).

Intracranial hemorrhage recurred in the two cases in which there were no or minimal differences in pressure.

3. We have found the ophthalmodynamometer to have its best application in the diagnosis of intermittent insufficiency of the internal carotid artery. As nearly as can be estimated, about 80 percent of such patients have a lowering of pressure in the retinal artery on the side of the carotid involvement. A significant lowering on the involved side is good evidence for such a diagnosis. Our neurologists rely very much on this measurement. Any patient who presents signs and symptoms of carotid insufficiency and who has unilateral lowering of the retinal arterial pressure is treated for carotid insufficiency without further ado. We rarely employ angiography under such circumstances, as we believe there is danger of converting carotid insufficiency into carotid thrombosis in the course of that process.

These studies have uncovered some heretofore unknown information about amaurosis fugax. This syndrome seems, at least in some cases, to be caused by some form of intermittent obstruction in the ophthalmic artery. Among 14 patients with thrombosis of the internal carotid artery, one patient gave a history of transient unilateral loss of vision. This continued four months, and then right hemiplegia and right homonymous heminopsia developed suddenly. However, among 38 patients with intermittent insufficiency of the internal carotid artery, 25 had a history of amaurosis fugax. Eight of these had cotton-wool patches in the retina on the same side.

We have had four patients whose only symptom was amaurosis fugax; administration of bishydroxycoumarin (Dicumarol) stopped the attacks. Two of these had lower pressures in the retinal artery on the affected side, but two had equal pressures on the two sides. One patient had noted from three to six complete blackouts in the left eye daily for nearly three weeks. Retinal arterial pressures measured 30 mm. Hg in each eye. He had such an attack while in the office. The pupillary light reflex was absent, and the eye was completely sightless. However, ophthalmoscopic examination showed no changes in the fundus of this eye. The arterial circulation of the eye collapsed completely at the merest touch with the instrument, indicating that even the systolic pressure was below the intraocular pressure. The pressure in the opposite eye went up to 40 mm. Hg. The patient had an immediate cessation of his attacks when administration of anticoagulants was begun. This information about amaurosis fugax opens up an intriguing field of inquiry.

Dr. Wood's citation of the 79-year-old woman with right hemiplegia and pressure of 90 mm. Hg in the right eye and 75 mm. Hg in the left which came down to 40 and 42 mm. Hg is interesting; so is that of the man who reported with dizziness and ataxia as well as tremendously elevated retinal arterial pressures. I suspect both of these cases, particularly the second, were instances of basilar arterial insufficiency, which sometimes results in such massive elevation of the carotid pressures as were noted. We have seen pressures drop precipitously several days or weeks after the stroke.

There is no question but that the retinal arterial pressures are elevated—sometimes extremely elevated—in the presence of increased intracranial pressure, and that they are lowered as soon as visible papilledema appears. However, there are possibilities for mistaken diagnosis here which I think almost too treacherous to permit use of the ophthalmodynamometer as a tool for diagnosing increased intracranial pressure when there

is no papilledema. The first of these is occlusive disease of the basilar artery, in which we have found a markedly elevated retinal arterial pressure among more than half the cases; and the second is generalized elevation of the blood pressure, which produces an extremely variable retinal arterial pressure.

In several patients with definite symptoms of insufficiency of the internal carotid artery we have found the lowering of pressure on the "wrong side." One of these on angiography was found to have bilateral occlusion even though the symptoms were unilateral.

In pulseless disease (Takayasu's syndrome) the retinal arterial pressure may be so low that spontaneous pulsation of the retinal artery is visible. We have observed this also among patients with postural hypotension; in such patients the retinal arterial pressures may be normal when the patient is supine, and yet may be too low to measure or even may give place to spontaneous pulsation when the patient stands.

Again I wish to thank Dr. Wood for his interesting paper and your society for allowing me to comment on this subject.

David Shoch,
Corresponding Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

January 17, 1957

Dr. I. S. Tassman, *Chairman*

Mandibulo-facial dysostosis (Treacher Collins syndrome)

Dr. Leonard Apt (by invitation): Mandibulo-facial dysostosis is a congenital deformity of the facial bones which has not been described often in the American literature. Although the key manifestations involve the eye region, the syndrome is little known to the ophthalmologist in this country. A total of 121 cases was found in 69 articles up to part of 1956; 16 cases described in eight papers were reported from the United States (only two reports were by American ophthalmologists). Dr. Apt has seen four patients with this entity in less than one year at the Wills Eye Hospital, and previously has encountered five cases while at a children's hospital. This experience suggests that the disease is more common than one would surmise.

The major clinical manifestations in mandibulo-facial dysostosis are:

1. Eye. The palpebral fissures slope downward laterally (antimongoloid); coloboma in the outer portion of the lower eyelids; deficiency of cilia in the medial half of the lower eyelids; occasionally absence of the puncta of the lower eyelids and abnormalities of the nasolacrimal channels.

2. Facial bones. Underdevelopment of the malar, maxillary, and mandibular bones; macrostomia; fistulas or clefts between the angles of the mouth and the ears.

3. Ears. Malformation of the external ear, auditory canal, and middle ear (incus and malleus).

The appearance of these patients with the fully developed syndrome is that of a fish or a bird. This is particularly unfortunate because most of the patients are of normal intelligence. Persons with the incomplete syndrome—those with no gross abnormalities of the lower face or ears—may go unrecognized.

The facial malformations have been explained by a failure of differentiation of the mesodermally derived tissue of the first visceral arch at about the seventh week of fetal life. The absence of the stapedial artery, a vessel which is associated with the first visceral arch, recently has been offered as an explanation for the maldevelopment.

Three points are to be stressed in the practical management of these patients:

1. Early detection and treatment of deafness to allow normal mental and social de-

velopment of the child. Deafness is a common feature of this syndrome and may not be suspected if there is middle ear but no external ear abnormality.

2. Investigation of the inheritance pattern by careful family history and by radiographic examination of the facial bones. There may be roentgenographic evidence of underdevelopment of the facial bones in individuals who have no gross facial abnormalities.

3. The necessity of good supervision and close co-operation between ophthalmologist and general plastic surgeon to obtain optimal cosmetic results. Complications due to congenital obstruction in the lacrimal drainage system are handled in the usual way by the ophthalmologist.

Comparative studies using primacaine HCl and other local anesthetics

Dr. H. G. Scheie, and Dr. Neil S. Williams (by invitation) reported further studies which attempted to find a local anesthetic of sufficient duration to avoid the period of immediate postoperative pain following ocular surgery. Primacaine HCl was made available on an experimental basis in June, 1956 (Novocol Chemical Manufacturing Company, Inc., 2911 Atlantic Avenue, Brooklyn 7, New York). Studies reported in this paper show that it has a duration of approximately one hour longer than procaine HCl or Lidocaine HCl. It is an excellent anesthetic agent, causes no local irritation, and seems nontoxic. It is being extensively used for dental surgery where it is considered to be the anesthetic agent of choice by many. The ideal anesthetic solution for ophthalmology surgery should, however, have a considerably longer action to prevent postoperative pain.

The authors also reported further experience with retrobulbar anesthesia using a polyethylene tube retained in the muscle cone. It has proved to be effective in relieving postoperative pain and has been used with safety in over 600 patients.

The pupil

Dr. Michael Curcio (by invitation): In clinical practice the pupil of the eye as it varies from patient to patient (and in the same patient from time to time) in size, shape, or position produces characteristic subjective visual effects.

By far the most important pupil characteristic is that of size. Practical observations, supported by theory, show that size changes have clinically significant effects upon retinal image brightness and depth of focus. The latter further influences accommodative demand for near vision, the age of onset of the presbyopic syndrome, and the tolerability of refractive errors, even in younger subjects.

In addition effects upon adaptation time, subjective appreciation of small opacities, and extent of visual fields were reviewed.

Special observations upon the results of anisocoria were discussed together with some original suggestions for its management.

William E. Krewson, 3rd,
Clerk.

PITTSBURGH SOCIETY OF OPHTHALMOLOGY

December, 1956

Dr. Charles Weisser, *President*

Myasthenia gravis

Dr. George Wright, Jr., reviewed the clinical aspects of myasthenia gravis. The movie, produced by the Myasthenia Gravis Foundation, was presented. Following the movie, Dr. George Wright presented a series of cases.

The first was that of a 57-year-old roll turner in a steel mill. The patient complained of tiring easily and difficulty in chewing for any length of time. The symptoms were improved with prostigmin. However, a lymphadenopathy and a ptosis of the left upper lid developed. Biopsy showed the pres-

ence of a lymphatic leukemia which aggravated symptoms of a mild myasthenia gravis.

The second case was one in which the diagnosis of multiple sclerosis had been made because of paresthesias and other findings. This diagnosis had been established for 10 years. A new syndrome of symptoms appeared in which there was ptosis, diplopia, and extreme fatigue.

The third case was one in which there was diplopia, rapid fatigue, and a total third-nerve palsy. Following the injection of prostigmin there was no improvement of the muscle palsy although there was definite improvement of other findings.

The last case was one which was characterized by an acute onset of fever, ptosis, and other findings which were originally considered as due to encephalitis but subsequently proved to be a case of myasthenia gravis of sudden onset.

Dr. Wright summarized his discussion by stating that 75 percent of all cases have eye symptoms; that more cases would be diagnosed earlier if the disorder were suspected; that findings might be detected late in the day which would not be present earlier in the day; and that, frequently, myasthenia was precipitated by emotional stress. Dr. Wright described the technique of the test using prostigmin five minutes after a hypodermic of 0.02 gr. atropine. Tensilon may be employed intravenously.

Sol Goldberg,
Reporter.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

November 5, 1956

Dr. Max Chamlin, *President*

Visual field defects due to optic-nerve compression by mass lesion

Dr. Max Chamlin presented the visual field findings in 23 cases of mass lesions involving the optic nerves but, as far as could be determined, not extending to the chiasm; 14 cases of sphenoid ridge meningioma; five cases of olfactory groove meningioma; three cases of orbital tumor; and one case of frontal lobe tumor. Early, as well as late, involvement of the visual fields was demonstrated. In all cases the periphery was involved before the central area. When the central area was affected, it was only a direct extension of the peripheral defect. In no case was a discrete "central scotoma" found —that is, without "break-through" to the periphery.

The use of appropriately small visual angles to detect early peripheral defects was stressed. In tumors involving one nerve, contralateral involvement usually manifested itself as a peripheral indentation in the temporal field of the contralateral eye.

Chiasmal lesions were excluded intentionally. It was pointed out that even in chiasmal lesions, early defect was found in the "intermediate" field, and not as a true "central scotoma." The hemianopic features of chiasmal lesions serve to identify the site of interference. Cases with papilledema were also excluded, since the enlarged blindspot may extend to the fixation area to simulate a central scotoma.

The differential diagnosis between optic atrophy due to optic neuritis and mass compressions was discussed. Some nerve-fiber bundle defects of optic neuritis were shown to resemble the altitudinal types of defect due to mass lesions. However, in many cases, the "sloping edge" of the mass lesion defect helped to distinguish it from the "steep edge" that may be found with the nerve fiber defect of optic neuritis. The great importance of a thorough, painstaking history in making such differential diagnosis was stressed.

Discussion. Dr. Kestenbaum said that the demonstrated cases were very interesting since they suggest the possibility of peripheral field defects without a central scotoma in tumors pressing on the optic nerve. One would, however, go too far if one would

deny the occurrence of central scotoma in pressure upon the optic nerve. According to the experience of Cushing, Foster Kennedy, and others, and my own experiences, central scotoma is the typical defect in involvement of the optic nerve by tumors. In chiasmal lesions, the presence or absence of a central scotoma is important in topical diagnosis. If a lesion, tumor, or arachnoiditis, or aneurysm of an anterior artery, affects the chiasm from in front, it must also affect at least one of the optic nerves; such an involvement is characterized by loss of the central vision (central scotoma) of the one eye. If a lesion affects the chiasm from behind, there is no central scotoma, the central visual acuity remains more or less intact.

DR. CHAMLIN said that it must be difficult simply to throw aside old doctrines and adopt new ones. His case reports, as well as those he quoted from the literature, leave little doubt about the frequency of peripheral defects without central involvement. He believes that the discrete central scotoma in mass compression is far less frequent than the literature would have us suppose. As for chiasmal lesions, he always found central involvement to occur as an extension of intermediate field defect, but the sharp hemianoptic features serve so well to identify the site of interference that confusion is not likely.

DR. GOODSIDE, who had noted bilateral involvement in several cases of olfactory groove meningioma, asked how this can occur without chiasmal involvement.

DR. CHAMLIN replied that this can occur when the lesion is anterior and extends back just beyond the optic foramina, without reaching the chiasm.

DR. POSNER commented that, although quite ignorant in matters of neuro-ophthalmology, he has been interested in visual fields in glaucoma. What is commonly referred to as a nerve-fiber bundle defect, such as the one demonstrated in two cases of optic neuritis, is more likely to bear a relationship to the vascular supply of the retina. The distribution of these scotomas seems more likely to correspond to the retinal blood vessels than to the nerve fibers. The nerve fibers which stem from the vicinity of the optic nerve are not the same as those which originate from the more distal parts of the retina, specifically from the temporal quadrant. Consequently, the nerve fibers which correspond to the nasal quadrant of the field are not the same as those which supply the temporal quadrant of the field near the blindspot. The fact that the defect is continuous tends to suggest a vascular origin. Furthermore, we find very similar defects in certain types of choroiditis, such as Jensen's juxtapapillary choroiditis, and in closure of a branch of the central retinal artery. It is very difficult to differentiate by means of the field between an inflammatory process and pathologic changes in the nerve fibers. This should be borne in mind in interpreting cases which present this type of field defect.

OPHTHALMOLOGY DURING THE MIDDLE AGES

HARRY SOBOTKA, PH.D., presented a paper that was published in the *Archives of Ophthalmology*, March, 1957, page 366.

Jesse M. Levitt,
Recording Secretary.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

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THE 1957 ACADEMY MEETING

The 62nd annual session of the American Academy of Ophthalmology and Otolaryngology was held in the Palmer House, Chicago, from October 13 to 18, 1957. Approximately 5,800 members and guests were registered. Many of these were from foreign lands. The weather was delightfully warm and sunny and remained so throughout the week of the meeting, although many members did not get outdoors at any time to enjoy it, for, as usual, activities of a social and scientific nature kept one busy from early morning until late at night.

The president, Erling W. Hansen of Minneapolis, called the joint session to order promptly at nine-thirty Monday morning, October 14th. The guest-of-honor was

Henry P. Wagener of Rochester, Minnesota, internationally known for his life work on ophthalmoscopy and particularly for his contributions on vascular diseases of the retina. The subject of his address was "Spasm and organic ocular disease," in which he presented considerable and logical evidence for the theory of retinal angiospasm.

The president spoke on the subject of "Looking backward—and forward," in which he discussed the history of the academy in a lively fashion. The development of the academy into an extraordinarily good educational institution was carefully noted. The present organization is indeed a most efficient and deeply appreciated one and we can only marvel at its existence. Twenty-two members were then awarded honor keys and certificates for their valuable work as instructors and for their contributions to the success of the academy.

Then followed a symposium on "Viruses and virus diseases." "The nature of viruses" was described by Jerome T. Syverton, professor of bacteriology and immunology, University of Minnesota Medical School. He gave us up-to-date information regarding these villians and fascinated us with a description of how they are grown on living cells and identified. Edwin H. Lennette, chief, Viral and Rickettsial Disease Laboratory, California Department of Public Health, spoke on "Viral infection of the upper respiratory tract," in which he described a number of the more common viral entities, lavishly illustrated with charts and diagrams. The symposium concluded with a paper on "Viral infections of the eye and adnexa," by Phillips Thygeson of San Francisco, widely known authority on the subject.

On October 15th, Girard, Fountain, Moore, and Thomas, all of Houston, Texas, gave a clinicopathologic case report, "Teratoma of the orbit." There followed a most informative symposium on the "Diagnosis and management of intraocular melanomas." Edwin B. Dunphy was moderator. The members were Arnold W. Forrest of White Plains, New York; Irving H. Leopold of Philadelphia; Algernon B. Reese of New York; and Lorenz E. Zimmerman of Washington, D.C. These experts covered the difficult subject most adequately in evaluating and summarizing our information regarding melanoma oculi up to the present. It will be a most valuable contribution when printed in the *Transactions* of the academy.

Theodore F. Schlaegel, Jr., of Indianapolis gave a paper on "Granulomatous uveitis: An etiologic study of 100 cases." He found that 18 percent of his cases had granulomatous uveitis involving the anterior segment alone. Investigation of causation included the usual tests. The results, also as usual, were disappointing. The Isoniazid therapeutic test for tuberculosis and the penicillin test for syphilis were found to be of value. Not a single response to Daraprim, when used as a therapeutic test for toxoplasmosis, was obtained.

Guillermo Pico of Puerto Rico showed an excellent motion picture of dacryocystorhinostomy, most skillfully performed, in which he emphasized the important part the use of the Stryker saw played in decreasing the amount of bleeding, so common in the operation.

Harold G. Scheie of Philadelphia described a new operation for glaucoma in his paper, "Retraction of scleral wound edges as a fistulizing procedure for glaucoma." A fistula was produced by superficial application of the actual cautery to the sclera adjacent to the lips of the wound just after completion of an ab externo incision, followed by a peripheral iridectomy. He showed many slides illustrating most excellent results from this operation, and a short motion picture illustrating his technique which appeared to be simple to execute. His paper was impressive.

The final event of the morning was the showing of a film of Meyer-Schwickerath of Bonn, Germany, on "Light-coagulation of retinal lesions," kindly sent over to the

meeting by its author. The members of the academy were most appreciative of this courtesy for most all of us had heard about this new and important contribution to our science but few had had the opportunity to get first-hand information. A commentary during the showing of the film was given by Derrick Vail who had had the opportunity of seeing the film and hearing Meyer-Schwickerath at the Oxford Congress in July.

The scientific program continued the next morning with a clinicopathology report on "Multiple cysts of the iris and ciliary body," by Torrence A. Mackley, Jr., of Columbus, Ohio. There followed the XIV Jackson Memorial Lecture sponsored by THE AMERICAN JOURNAL OF OPHTHALMOLOGY, given by Michael J. Hogan of San Francisco whose topic was "Ocular toxoplasmosis," a subject in which he is a recognized authority. It was beautifully presented and illustrated. As is customary, the lecture will be published in THE JOURNAL.

Brian J. Curtin and C. C. Teng of New York discussed the "Scleral changes in pathologic myopia," a subject that has been quite neglected in our literature. They found posterior thinning and special degenerative changes in the scleral fiber bundles, mainly in collagen and elastic tissue.

William E. Borley and Alan A. Snyder of San Francisco showed an exciting motion picture on "The surgical treatment of high myopia: Combined lamellar scleral resection with scleral re-enforcement using a donor eye." They reported the successful use of this procedure in nine cases in patients from the age of three years to middle age, with an average reduction of 7.2 diopters. The myoptic degenerative changes are halted and there is usually a visual improvement.

"The requisites for postoperative third-degree fusion" were given by Raynold N. Berke of Hackensack, New Jersey. He made a most detailed clinical survey of 256 cases of squint and showed that the prognosis for a functional cure in constant squint was always poor when the squint began at birth, was of long duration, the vision in the amblyopic eye 20/50 or worse, or when the residual deviation was over 10 prism diopters. On the other hand, the prognosis was always good in intermittent squint, when the vision was good in each eye, and the deviation was fully, or almost fully, corrected.

Following this excellent paper, Olga Ferrer of Havana, Cuba, showed a fine motion picture of the insertion of a Strampelli plastic lens in the anterior chamber.

"The pathology of the trabecular meshwork in open-angle glaucoma" was described by Milton Flocks of San Francisco, who had the rare opportunity of examining eyes of this disease uncomplicated by late changes, such as synechias. He described marked degenerative changes in the trabecular meshwork, especially along the inner wall of Schlemm's canal. He found essentially the same changes in the trabecular meshwork of eyes with secondary glaucoma with open angles and concluded that these changes were secondary to the increased intraocular pressure and not the cause of the increased pressure. His theory was not acceptable to Herbert M. Katzin of New York and Georgiana Dvorak-Theobald of Oak Park who were the formal discussors.

On Thursday, October 17th, the scientific program consisted of a clinincopathologic report on "Eosinophilic granuloma of bone" by Bradley R. Straatsma of New York. Then followed a presentation of new instruments, following which Brittain F. Payne and John T. Simonton of New York showed a motion picture of "Lateral orbitotomy for retrobulbar film." It demonstrated the effectiveness of the use of the Berke lateral incision and the Stryker saw. The operation was neatly performed.

The next event was the long-awaited one of a symposium on "Scleral resection procedures." It was moderated by Dohrmann K. Pischel of San Francisco, who gave the introduction, indications, and, at the end, the conclusions. Graham Clark of New York

discussed the mechanical factors. Peter C. Kronfeld of Chicago described the current techniques. Charles L. Schepens of Boston described his scleral buckling operation, and P. Robb McDonald of Philadelphia, the complications of scleral resection and buckling procedures. The majority of the members of the panel believed that there was always a certain amount of buckling present after any type of resection. Schepens was in the minority in this belief. All authors were firm in the necessity for accurately localizing the breaks or tears in the retina and sealing them off, regardless of the type of surgery used.

A. Edward Maumenee, Baltimore, showed a most excellent and exciting motion picture of his method of surgical removal of epithelial downgrowth from the anterior chamber. There was prolonged applause from the crowded audience.

G. LeRoy Porter of Urbana, Illinois, then gave a paper on the "Incidence and detection of glaucoma by routine tonometry." He made routine tonometric measurements in 2,000 patients over 40 years of age and found that a substantial number of glaucoma cases which would otherwise have passed unrecognized were detected.

The final session, held on October 18th, was surprisingly well attended for it is notorious that many of the members have left the night before. Those who were there were well rewarded for their endurance. The clinicopathologic report was given by Alson E. Bradley of Iowa City, Iowa, on "A case of Behçet's disease."

John W. Henderson of Rochester, Minnesota, showed a film on "Tarsal-periosteal-frontalis sutures for correction of palpebral ptosis," which looks like a simple and effective method to try in all forms of ptosis. The basic aim is to suspend the tarsal plate of the eyelid from the periosteum along the superior rim of the orbit.

The puzzling problem of "Pseudotumor cerebri" was presented by J. Lawton Smith, neurosurgeon of Baltimore. The diagnosis of this condition is difficult and the treatment is controversial.

Lorenz E. Zimmerman of Washington, D.C., gave a paper of considerable interest on "Some applications of histochemical methods for the demonstration of acid mucopolysaccharides to ophthalmic pathology." He showed many beautiful slides of ocular specimens that clearly demonstrated that some acid mucopolysaccharides are hyaluronidase-sensitive, for example in the cornea as compared with the sclera.

Algernon B. Reese and Frank D. Carroll in a paper, "Optic neuritis following cataract extraction," presented definite evidence that this is one of the entities responsible in a few cases for decrease of vision occurring some time after cataract extraction. They reported 17 cases of this syndrome, three of which were bilateral. They differentiated this condition from poor vision following the operation due to ocular hypotony, uveitis, retrobulbar injections, and macular edema with posterior separation of the vitreous.

Daniel Snydacker reported on "Diabetic neuropathy as a cause of extraocular muscle palsy," and clarified for us many of the salient points of this condition.

The final paper, "Ophthalmologic allergy," was by Alson E. Braley of Iowa, which consisted of a recapitulation of our knowledge of this condition. His talk was sponsored by the American Society of Ophthalmologic and Otolaryngologic Allergy.

The only criticism that I heard of the scientific sessions was that they were so interesting you couldn't fall asleep.

There were, as usual, special scientific programs provided by the Teachers' Section, the American Orthoptic Council, the American Association of Orthoptic Technicians, the Committee on Plastic and Reconstructive Surgery, and the American Society of Ophthalmologic and Otolaryngologic Allergy.

There were 14 scientific exhibits in ophthalmology of high excellence. Milton Flocks and Homer Brugge of California received the first award for their exhibit "Studies of

the trabecular meshwork of normal and glaucomatous eyes." Benjamin Milder and Byron H. Demorest of Saint Louis received the second prize for their exhibit "Dacryocystography," and Harold F. Falls, Burton R. James, and Frederick A. Waara, Ann Arbor, Michigan, were given the third award for the exhibit, "Acrylic schematic eyes."

On each afternoon, hours in instruction, consisting of 160 individual courses and 50 continuous ones, were given by a faculty comprised of 256 instructors. The hours of instruction totalled 405. The courses were crowded with eager audiences.

The social side of the meeting was far from neglected. The formal banquet on Monday night was a sell out for the members were jostlingly anxious to see and hear Dorothy Shay sing her famous and at times infamous hill-billy tunes and, following this, to dance to enticing music for several hours. The usual alumni functions were held on one of the evenings. This year, there were 34 of these dinners going on simultaneously in various hotels.

The Pan-American Association for Ophthalmology put on a gay fiesta which was well patronized and lots of fun. In addition, there were innumerable extracurricular cocktail parties and room pub-crawlings in between times. The sputnick was not even mentioned. There surely seemed to be more than 24 hours in each day.

The officers for 1958 are LeRoy Schall of Boston, president; John H. Dunnington of New York, president elect; F. Bruce Fralick of Ann Arbor, Michigan, first vice-president; David D. DeWeese, second vice-president; and Philip M. Lewis of Memphis, Tennessee, third vice-president. Victor Goodhill of Los Angeles is the new councillor.

The next meeting (1958) will be held in Chicago at the Palmer House, October 12th to 17th.

Derrick Vail.

OBITUARY

W. Gordon M. Byers, M. D.
1872-1957

William Gordon Matthew Byers died on August 2, 1957, at "Home Acre," Knowlton, P.Q., where he had taken up residence on his retirement from practice in 1946. With his death passed a man who had been at the heart and inspiration of ophthalmology in Canada, perhaps more than any other. The reasons for this lay not only in his personal character and many talents but also in the fact that his professional career straddled a most actively formative period in medicine and ophthalmology. He had a formidably exact and methodical mind. This was no better seen than in the manner in which he developed his literary scientific presentations. Each sentence was worked over and over again, often to the exasperation of his assistants, in order to obtain his exact meaning as precisely and briefly as possible. The result was that his papers were models of scientific literary style. They numbered over 40, most of them on pathologic or clinical subjects in which he was particularly interested.

The same punctiliousness and self-discipline marked his attitude to the practice of his profession. The clinical investigation of his patients was planned and executed to the last detail. Woe betide the houseman, or nurse, who failed to reach his exacting standards in the ward or in the operating room. He was a most skilled surgeon, particularly in intraocular surgery. As a result, he was held in immense respect by his patients, confreres, and juniors alike.

Though there was a certain egocentricity, a certain stiffness of manner and sterness of character, a degree of rigid rectitude about Dr. Byers, he always was fair and kind, sometimes extraordinarily so. With the passing years, this kindness and generosity dominated his character.

His ambition, or perhaps better stated his vision, was to stimulate, guide, or even drive if need be, the development of ophthal-

mology, first in his native city of Montreal and later in Canada as a whole, along the lines of highest endeavor and ethical standards. Being one of those referred to by Prof. Arnold Toynbee as the "dominant minority," and a man of the highest personal integrity and ethics, this is the only way it could be. In this endeavor he largely succeeded but only at the expense of enormous effort. Ophthalmology in Canada and Montreal today owes him a great debt.

Dr. Byers was born in Gananoque, Ontario, on December 17, 1872. He was graduated in medicine from McGill in 1894 with first class honors. During the next two years he interned in medicine and surgery at the Montreal General Hospital. Then he studied in Edinburgh, obtaining the L.C.P. and S. Following this, he served for 18 months as house surgeon at the Royal London Ophthalmic Hospital and later he studied for a year in Berlin, Paris, Vienna, and Utrecht. Dr. Byers was always an assiduous student. His enthusiam carried him into many new fields in ophthalmology. As a result of his postgraduate studies and scientific contributions he earned a D.Sc. from McGill University in 1909. This degree was the highest scientific accolade at that university.

On his return from Europe in 1898, he joined the staff of the Royal Victoria Hospital, Montreal, and a year later that of McGill in the Department of Ophthalmology. He became ophthalmologist-in-chief of the first in 1921, and professor and chairman of the department of the latter in 1923. On reaching the respective retirement ages at each institution, he forsook the appointment of the first in 1935 and the latter in 1937. He carried on private practice for a few years until 1946, when he retired.

In the light of his character, training, and aims, it is not surprising that Dr. Byers took an active part in several ophthalmic societies. In 1920, he actively inspired the founding of the Montreal Ophthalmological Society. He was its first president. This society, embracing both the English- and French-speaking ophthalmologists of Montreal, has taken a very active part in the clinical life of the city ever since. Again in 1937, he was largely responsible for the founding of the Canadian Ophthalmological Society, which since has developed into the most important clinical, scientific, and even political group of ophthalmologists in Canada, and he was its first president. No better epitaph could be made to Dr. Byers than to say that these societies were so soundly conceived and founded that they have flourished with ever-increasing importance.

Dr. Byers also was an active member of the American Ophthalmological Society from his election in 1906, and was its president in 1934. He was also a member of the Ophthalmological Society of the United Kingdom from 1897 on.

All his life, Dr. Byers was a man of most studious and varied tastes. Nor did he confine himself to ophthalmology. Anything that attracted his interest, he attacked with consuming enthusiasm. It was thus that he approached his varied hobbies such as sailing, farming, gardening, and bird watching. His indefatigable companion and helpmate in all these activities was his wife, Jean Coates Newton, whom he married in 1911. She survives him, and to her and her family go our deepest sympathies. Of this union survive two sons, Donald and Malcolm, and two daughters, Mrs. John Lafleur and Miss Helen Byers. One son, Allan, was killed while serving with the R.C.A.F. in World War II.

John V. V. Nicholls.

CORRESPONDENCE

Ptosis and vertical muscle anomaly

Editor,
American Journal of Ophthalmology:

Regarding the correspondence on "Ptosis and vertical muscle anomaly," in the August, 1957, issue of The Journal, and the cases

presented by Dr. Warren Binion (Am. J Ophth., 43:630, 1957), as well as the cases of ptosis associated with a horizontal muscular defect described by Dr. Harris Tomkin in his letter (Am. J. Ophth., 44:280, 1957), I feel it should be put on record that Dr. Jorge Malbran of Buenos Aires was the first to stress the importance of correcting the ptosis of the lid (Strabismes et Paralysies, 1949).

(Signed) J. E. Wolff,
Johannesburg, South Africa.

BOOK REVIEWS

DORLAND'S ILLUSTRATED MEDICAL DICTIONARY. Edited by L. B. Arey, W. Burrows, J. P. Greenhill, and Richard M. Hewitt. Philadelphia and London, Saunders Company, 1957, edition 23. 1,598 pages, more than 700 illustrations and 50 plates. Price: $12.50.

This is perhaps the most familiar medical dictionary to the physician. The same rose-red cover can be recognized a mile away. The style and print is the same as usual so that it is like an old friend in a new suit who has become wiser and more comprehensive with each edition.

There is included, as an extra bonus, sections on "Modern drugs and dosages," by Austin Smith, the editor the the *Journal of the American Medical Association*, and "Fundamentals of medical etymology," by Lloyd W. Daly, associate professor of classical studies, University of Pennsylvania. These are most excellent.

The distinguished philological consultants are Paul J. Alexander, Ph.D., associate professor of history at Brandeis University, and Harry C. Messenger, M.D., formerly instructor in Greek and Latin at Harvard University and Radcliffe College.

Karl W. Ascher, M.D., of the University of Cincinnati, and Edwin F. Tait, M.D., of Temple University School of Medicine, are the consultants and major contributors in the field of ophthalmology.

The presence of these gentlemen and the eminent editorial board assures us of a first class piece of work and we are not disappointed.

Derrick Vail.

REGULATION AND MODE OF ACTION OF THYROID HORMONES. G. E. W. Wolstenholme and E. C. P. Millar, editors. Ciba Foundation, Colloquia on Endocrinology, Volume 10. Boston, Little, Brown and Co., 1957. 311 pages, 114 illustrations, author and subject indices. Price: $8.50.

The problem of the ocular complications of Graves' disease continues to plague the ophthalmologist and, although he does as well as he can to alleviate the threat to vision and discomfort that beset the patient with this disease, he knows that definitive treatment must await the elucidation of the basic etiology of hyperthyroidism.

This volume is the transcript of a conference held in England in June, 1956, which dealt with the basic problem of the regulation of the thyroid hormones. Most of the papers are quite technical but many interesting clinical points are raised. For example, Harris and Woods put forward the hypothesis that thyroid hyperactivity is caused by a lack of adrenal cortical response in times of stress. This is on the basis of experimental evidence which indicates that the corticosteroids exert an inhibitory effect on thyroid activity. The whole concept of the hypothalamus-pituitary-thyroid axis is discussed by Crown-Grant under the title "The 'feed-back' hypothesis of the control of thyroid function." The remaining 16 papers are primarily concerned with the chemistry and biochemistry of the thyroid hormones, and it is the aforementioned article which will interest the ophthalmologist who has made a special study of thyroid problems.

David Shoch.

EINFUEHRUNG IN DIE AUGENHEILKUNDE (Introduction of Ophthalmology). By Prof. Dr. P. A. Jaensch. Stuttgart, Georg Thieme Verlag, 1957. 248 pages, 197 illustrations, some in color. Price: DM 27.50.

The second edition of this useful little textbook was reviewed in this space (**37**:129, [Jan.] 1954). The appearance of a third edition only three years later is eloquent proof of its usefulness to the medical student and the general practitioner. A renewed acquaintance reaffirms the excellent general impression gained from the previous edition. This is particularly true for the presentation of the external diseases and the discussion of the visual field defects.

Although the title page claims that this is an "improved" edition, it should be stated in all fairness that the new edition is essentially unaltered. Only very few of the illustrations are different—mostly enlargements of those from the second edition or enlarged cut-outs that bring out the features to be illustrated in greater detail.

A few additions help to bring the text up to date. Briefly mentioned in the new edition are the acrylic lenticuli of the Ridley and Strampelli type. Diabetic retinopathy is discussed in greater detail. Diamox is included in the discussion of the medical treatment of glaucoma.

The author is correct in avoiding discussion of the treatment of strabismus, a field that should concern only the ophthalmologist. Apparently reviewers of the previous edition criticized this omission. But why avoid orthoptics, yet mention pleoptics, a topic that is much more controversial?

The failure to emphasize the difference between the serous and granulomatous type of anterior uveitis has already been singled out by this reviewer in the discussion of the second edition. Likewise, scleral resection and lens extraction as surgical treatment of myopia have been retained in the new edition as accepted forms of therapy.

Stefan Van Wien.

EXPERIMENTAL PSYCHOLOGY AND OTHER ESSAYS. By Ivan Petrovitch Pavlov. New York, Philosophical Library, 1957. 653 pages, no index. Price: $7.50.

Every student of medicine knows of Pavlov and at least some of his classical studies in physiology and psychiatry. This volume consists of translations of his significant works on the circulation of the blood, trophic action of the nervous system, digestion, laws of development, higher nervous activities, sleep, physiology and psychology, experimental pathology, and, most delightful of all, fragments of his statements at the famous "Wednesday" gatherings. There is a very short autobiography, too short for one's appetite.

The translations are well done, apparently, for the English style flows along without being awkward. Those who are interested in a study of this great man will find this book exceedingly worth while.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisiions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

9

GLAUCOMA AND OCULAR TENSION

Otto, Joachim. **The loss of the fixation point after antiglaucomatous operations.** Klin. Monatsbl. f. Augenh. 131:178-195, 1957.

In the first part of this report the results after a trephine operation are analyzed. In 336 eyes the operation was done when the fixation point was already endangered. In 12 of these eyes central vision was lost. This occurred in only two eyes immediately after the operation, in the others is happened one or several years later. Half of these patients had a hypotonic eye, in the other half the tension was normalized. Out of 891 eyes with only mild field defects three lost central vision after the operation. This loss was caused by a vascular lesion (thrombosis, spasm) in two, and by a disciform macular degeneration in one eye.

In the second part the results after cyclodialysis are analyzed. Out of 387 eyes with a large field defect approaching the fixation point central vision was lost postoperatively in only three. This occurred 2, 10 and 12 years after the operation. No loss of central vision occurred in 516 eyes with only mild field defects.

The postoperative loss of central vision is so rare that its possible occurrence should not be a contraindication to the operation. (6 figures, 2 tables, 90 references) Fredrick C. Blodi.

Pommier, L. and Etienne, R. **Glaucoma.** J. de méd. de Lyon 899:603-608, June 20, 1957.

The authors point out how important it is that glaucoma should be understood and recognized by all physicians. To this end they provide an adequate exposition. F. H. Haessler.

Rosengren, B. **Rise in the ocular tension produced by circumlimbal pressure on the sclera.** Tr. Ophth. Soc. U. Kingdom 76:65-72, 1956.

Whether the inflow of aqueous is increased or the outflow is obstructed in the different types of glaucoma is a question discussed by the author. He used a suction cup placed on the perilimbal area. A negative pressure of 50 mm. Hg produced a rise in tension after application from five to 30 minutes. The average rise in tension was 15.5 mm. After removing the suction cup in normal eyes the ten-

sion returned to normal after 10 to 30 minutes. The time in glaucoma simplex was 35 to 70 minutes. (6 figures, 9 references)

Beulah Cushman.

Schmerl, E. **Contribution to the problem of ocular tension.** Ann. d'ocul. **189**: 901-922, Nov., 1956.

The author presents his theories on the central control of ocular tension. In section I he gives evidence for the existence of such centers. For example he feels that the water-drinking test is dependant on the water-regulating function of the hypothalamus and pituitary. He states that administration of barbiturates depresses the pituitary and therefore interferes with the hydremia test. In section II he describes the action of light and darkness on ocular tension and their relationship to the two pituitary factors he has found in the spinal fluid. These he calls hyperpiesin and myopiesin. In section III the author records the results of stimulation or ablation of the ciliary ganglion and the superior cervical ganglion and in section IV applies these experimental findings to the problem of the pathogenesis of glaucoma. (1 table, 25 references)

David Shoch.

Schmidt, Theo. **Applanation tonometry.** Ophthalmologica **133**:337-343, April-May, 1957.

Goldman demonstrated the first model of his applanation tonometer before the French Ophthalmology Society in 1954. In the present report a slightly modified instrument and its practical use are described. The effect of corneal astigmatism upon the tonometric reading is discussed.

On four fresh enucleated human eyes good agreement was obtained between manometric and tonometric measurement of the intraocular pressure. (3 figures, 2 tables, 1 reference)

Peter C. Kronfeld.

Schmidt, Theo. **The "differential value" as a measure of ocular rigidity.** Klin. Monatsbl. f. Augenh. **131**:195-202, 1957.

The differential value is the difference between the P_o in average rigidity and the P_o in the actually present rigidity. This value is nearly constant for a certain rigidity and seems to be a function thereof between a pressure of 8 and 55 mm. Hg. This value is positive in increased rigidity and vice versa.

The actual P_o can be determined with the applanation tonometer. A second value is then obtained with the 7.5 gm. weight on the Schiøtz tonometer and converted into mm. Hg on the 1955 Friedenwald table which uses the average rigidity coefficient of 0.0215 as basis. The difference of the two values is the "differential value" and this corresponds to only one rigidity factor which can be found on a conversion table.

The determination of this value is necessary for tonometry on patients who can not be brought to the slitlamp or other nonportable instrument. If an applanation tonometer is not available repeated paired readings can be used. (4 figures, 3 tables, 5 references)

Frederick C. Blodi.

Stepanik, J. **The episcleral venous pressure in eyes with low ocular tension.** Ophthalmologica **133**:397-406, June, 1957.

The paper concerns itself with the aqueous dynamics in eyes with ocular tension of 10 mm. or less. In 12 such eyes the author measured the episcleral venous pressure and arrived at a mean of 10.1 mm., the same as in normal eyes. These hypotonic eyes, very characteristically, did not show any aqueous veins by slitlamp examination. The aqueous, therefore, must have drained into other channels. There were signs of fistulation into the conjunctival sac, through traumatic or operative fistulas, in seven

of the twelve cases. For the estimation of the flowrate of such eyes the author recommends Grant's original formula $F = C \times (P_o - 4)$.

The estimates for the ocular tension in very hypotonic eyes may be inaccurate since in the calibration process the possibility of deformation of the globe by the extraocular muscles has not been taken into consideration. (1 figure, 22 references) Peter C. Kronfeld.

Streiff, E. B., Cuendet, J. F. and Cohen, R. **Geographic, chronologic and genealogic study of infantile glaucoma.** Ophthalmologica 133:342-345, April-May, 1957.

A series of 54 cases of congenital glaucoma was reviewed. In 17 percent of the cases more than one sibling was affected. There was a high incidence of associated ocular or extraocular congenital anomalies. Only meager evidence was disclosed of significant illness of the mother during the pregnancy. Peter C. Kronfeld.

Tornquist, Ragnar. **Corneal radius in primary acute glaucoma.** Brit. J. Ophth. 41:421-424, July, 1957.

There have been no previous studies of possible changes in corneal radius during glaucoma and in this study a comparison is made between radius of curvature of the cornea in acute glaucoma as compared to that of the cornea in the normal eye. The Javal-Schiøtz ophthalmometer was used for the measurements. In a representative mixture as to age, sex and refractive error, the chamber depths and the corneal radius were measured in 146 eyes of 95 patients with acute glaucoma and similar measurements were made in a series of 398 normal eyes. The mean average of radius in the normal was found to be 7.78 while in the glaucomatous eye it was found to be 7.53, a difference of 0.25 which is of sufficient amount to be significant. Tornquist believes that the small radius of curvature disposed to the disease and not that the disease resulted in the smaller curvature. (1 figure, 3 tables, 10 references). Morris Kaplan.

Ustinova, E. **The water-drinking campimetric test in the early diagnosis of glaucoma.** Vestnik oftal. 4:41-45, July-Aug., 1957.

Five groups of patients, 50 in each group, were studied by the author. The first control group consisted of patients in good general health, who had various eye diseases; in the second group the patients had glaucoma at various stages and 30 of these patients had cardiovascular disease. In the third group the patients had glaucoma in one eye only and 27 were affected with cardiovascular disease; in the fourth group glaucoma was suspected and in the fifth group the patients chiefly had hypertension with cardiac disturbances.

The campimetric test was done in a darkroom. The blind spot was examined before, every 15 minutes during the first hour and every 30 minutes during the next three hours. The test was considered positive if after the drinking of 200 or 500 cc. of water the blind spot was enlarged over five degrees in the vertical diameter and more than 33 percent in the horizontal diameter. The test was positive in 96 percent of the patients with glaucomatous eyes, in 90 percent of the patients with glaucoma in one eye, in 70.8 percent of the eyes in which glaucoma was suspected; of this latter group the diagnosis was confirmed clinically in 50.8 percent of the eyes. In the patients with cardiovascular disease the blind spot was not enlarged. The influence of the water-drinking test on the arterial pressure showed no appreciable change in the majority of the patients. The test can be used safely on elderly patients.

The maximal enlargement of the blind spot was observed an hour after the test

and the return to its normal size was usually between two and three hours after drinking the water. The sensitivity of the test was but slightly different after the use of 200 or 500 cc. of water; either quantity could be used effectively. Some methods were also used for comparison: the diurnal change of the intraocular pressure, Filatov-Kalfa's elastotonometry and Zeidel's dark tonometric test.

The author states that the water-drinking compimetric test is highly sensitive and is one of the best methods in the early diagnosis of glaucoma. However, none of the tests are infallible; in eyes suspected of having glaucoma various tests must be used for a correct diagnosis. (2 tables) Olga Sitchevska.

Weekers, R. and Watillon, M. **Congenital glaucoma without enlargement of the cornea.** Ophthalmologica **133**:37-45, Jan., 1957.

Four cases of glaucoma in young adults had in common, 1. apparently congenital anomalies of the chamber angle, 2. corneas of normal size, and 3. gradual onset of the glaucoma during the second decade of life. The chamber angle anomaly is described as mesodermal tissue partly filling the deepest portion of the angle. These cases seem to represent a rare, but definite type. The glaucoma may be due to blocking of the trabecular spaces by pigment particles which were part of the mesodermal tissue. Iridencleisis proved to be effective therapy. (4 figures, 6 references) Peter C. Kronfeld.

Yeroshewsky, T. **Medical and surgical treatment of glaucoma.** Vestnik oftal. **4**: 7-23, July-Aug., 1957.

In this extensive review of the foreign literature the newer drugs for medical treatment of glaucoma (Diamox, Mintacol, Dibenamin) are discussed. The antiglaucomatous operations used by leading American, English, French, Italian, and Spanish surgeons are reviewed in detail with comments by Yeroshewsky. The filtering operations: iridencleisis, cyclodialysis, corneoscleral trephining, the techniques of various surgeons, the use of gonioscopy, the mode of action of cyclodiathermy, and the various methods of cauterization of the ciliary body are given. A large bibliography (two pages) is appended. Olga Sitchevska.

10

CRYSTALLINE LENS

Agarwal, L. P. and Malik, S. R. K. **Diamox in cataract surgery.** Ophthalmologica **133**:153-159, March, 1957.

The routine use of Diamox just before and for three days after cataract surgery in doses of 250 mg. twice a day is recommended because it seems to reduce the incidence of vitreous prolapse during the operation and of iris prolapse and delayed chamber restoration after the operation. (2 tables, 6 references) Peter C. Kronfeld.

Arkin, Wiktor. **Congenital ectopia of the lens.** Klinika Oczna **27**:169-173, 1957.

The syndrome of congenital ectopia of the lens was traced through four generations consisting of 39 individuals. Ectopia of the lens was present in 12 of them. In addition to dislocation of the lens there were symptoms of Marfan's disease in one branch of the family and of Marchesani's type in the other. There was no definite pattern of dominance. The author discusses theories of etiology and heredity of ectopia lentis. (1 figure) Sylvan Brandon.

Binkhorst, C. D. **The Ridley operation: results obtained in 12 cases.** Ophthalmologica **133**:384-392, June, 1957.

In this series of 12 cases were three strictly unilateral (traumatic) and eight predominantly unilateral (senile) cata-

racts. The postoperative reaction was limited in all cases to a very mild anterior uveitis subsiding within two to three weeks. The mildness of the postoperative reaction is attributed to 1. minimal operative trauma to the uvea, 2. thorough removal of lens remnants by irrigation, 3. thorough mechanical cleansing of the acrylic implant, and 4. its sterilization by ultraviolet light. The final vision was 20/60 or better in ten of the twelve cases. (2 figures, 18 references)

Peter C. Kronfeld.

Choyce, D. P. **Anterior dislocation of the lens in Marfan's syndrome.** Brit J. Ophth. 41:446-448, July, 1957.

A 14-year-old girl, in a family of which several members had Marfan's syndrome, presented most of the typical signs of the disease including dislocation of the lenses. With glasses for aphakia her vision was 6/6. One day while she was stooping one lens became dislocated forward and so remained. During an attempt to replace the lens, it was grasped and held by the pupillary sphincter. Acute glaucoma quickly supervened and the lens was then removed under general anesthesia. A lens that is dislocated forward should be removed without delay. (1 figure, 1 reference)

Morris Kaplan.

Landolt, E. **The so-called exfoliation of the anterior lens capsule.** Ophthalmologica 133:309-320, April-May, 1957.

Landolt has reviewed the histologic material upon which Vogt based his concept of capsular exfoliation, that is of a degenerative, exfoliative process of and within the lens capsule that gives rise to crumb-, flake- and felt-like fragments on the anterior surface of the lens, on the posterior surface and at the pupillary border of the iris and at other sites in the anterior segment. After elimination of those eyes in which there was a possibility of distortion or modification of the disease by other processes there remained 18 eyes of 11 patients with clear-cut histologic and, in many of them, detailed clinical findings. After careful study of this material Landolt arrives at the very significant conclusion that the concept of exfoliation of capsular material should be abandoned. Instead, the disease should be considered as a uveal of an unknown, but not capsular-material syndrome characterized 1. by deposits on all walls of the posterior chamber, 2. by an abnormal looseness of uveal pigment, 3. by breakdown of the vitreous and 4. by increased permeability of the anterior blood-aqueous barrier for fluorescein. (9 figures, 27 references)

Peter C. Kronfeld.

Verrey, Florian. **Congenital cataract.** Ophthalmologica 133:302-309, April-May, 1957.

This is an attempt to draw some general conclusions from a series of 89 cases of congenital cataract observed and treated at the University Eye Clinic of Zürich (Switzerland). A presumptive etiology (heredity, acute or chronic disease of the mother or exposure to X rays during pregnancy), was disclosed in 23 cases; in the remaining 66 there was no clue whatsoever to the etiology. Associated ocular anomalies were nystagmus (in one third of the cases), strabismus (also in one third) and microphthalmus (in one fourth). Very common was a state of general retardation. The neurologist found a high percentage of brain stem disorders. The associated ocular or other anomalies were significantly more frequent in Verrey's series than in the series from the Wilmer Institute (Owens and Hughes, Arch. Ophth. 39:339, 1948).

Regarding surgery, Verrey favors optical iridectomies of various forms for the central, nonprogressive cataracts. The over-all results of operations on the lens were not quite as good as those of the Wilmer Institute. This difference was

probably due to a larger admixture of initially (prior to surgery) poorer eyes in the Verrey series. That many eyes with congenital cataract are underdeveloped or damaged eyes in respects other than their crystalline lenses is probably the most important finding in this paper.

In the discussion, Franceschetti refers to the syndrome of mental retardation, cataract, glaucoma and insufficiency of the tubular apparatus of the kidneys causing hyperaminoaciduria (Lowe, Terry and MacLachlan, Am. J. Dis. Child. 83: 164, 1952). Seven out of 10 consecutive cases of congenital cataract showed this hyperaminoaciduria. The study will be continued. (14 references)

Peter C. Kronfeld.

Weisel, J. and Swan, K. C. **Mydriatic treatment of shallow chamber after cataract extraction.** A.M.A. Arch. Ophth. **58**: 126-129, July, 1957.

In 14 cases of loss of the anterior chamber after cataract extraction the authors felt that the most significant finding was pupillary block. The treatment was to produce extreme mydriasis. (6 references)

G. S. Tyner.

11
RETINA AND VITREOUS

Babel, J. **Retrolental fibroplasia in one twin.** Ophthalmologica **133**:295-298, April-May, 1957.

Of two nonidentical twins kept in one incubator at oxygen concentrations not exceeding 40 percent for 40 days, one developed severe retrolental fibroplasia while the other one is reported to have normal eyes. The literature on retinopathy of prematurity in twins is reviewed. (8 references)

Peter C. Kronfeld.

Danielson, Ralph W. **The differential diagnosis of retinoblastoma.** A.M.A. Arch. Ophth. **58**:15-18, July, 1957.

In this paper the author stresses the armamentarium and extent of examination as well as the appearance and salient facts which differentiate retinoblastoma from confusing entities. (9 references)

G. S. Tyner.

Davis, L. A. and Diamond, I. **Metastatic retinoblastoma as a cause of diffuse intracranial calcification.** Am. J. Roentgenol. **78**:437-439, Sept., 1957.

The authors describe a four-months-old infant with a rapidly enlarging head and abnormal ocular movements. The right eye gave a yellow cat's eye reflex and a retroretinal mass was found. Postmortem examination revealed a primary retinoblastoma of the right eye. The tumor extended posteriorly, destroyed the optic nerve, expanded the optic foramen to a diameter of 1 cm., completely destroyed the pituitary gland and invaded the brain. (8 figures, 7 references)

F. H. Haessler.

Dorello, Ugo. **Electrocardiographic findings in cases of retinal vein thrombosis.** Riv. oto-neuro-oftal. **37**:258-266, May-June, 1957.

Forty patients with retinal vein thrombosis were studied electrocardiographically. A degree of myocardial change was found which exceeded that expected in patients of this age group. In addition the circulatory status of the patients did not seem to warrant such a high incidence of myocardial changes. The author therefore suggests the hypothesis that some cases of retinal vein thrombosis can be related to a cardiac lesion. (1 table, 3 references)

William C. Caccamise.

Dufour, R. and Bianchi, G. **Retinal detachment and glaucoma.** Ophthalmologica **133**:241-247, April-May, 1957.

The authors report five cases of retinal detachment in eyes affected with congenital or chronic simple glaucoma. From the clinical study of their cases and others

reported in the literature they conclude that a retinal detachment in a glaucomatous eye is, as a rule, an idiopathic detachment. A state of hypotony, brought on by medical or surgical therapy, may play the role of a precipitating factor, but there is always the possibility of the detachment itself having caused the hypotony. A high ocular tension may protect an eye with retinal breaks against a detachment.

It is very important not to ignore the possibility of retinal detachment in patients with chronic glaucoma. Adrenergic stimulants permit safe dilation of the pupil and thorough fundus study. After a successful detachment operation one has to prepare oneself for a return of the ocular tension to its level before detachment. Successful treatment of the glaucoma, medically or surgically, does not entail the danger of a recurrence of the detachment as long as the retina remains "watertight."

In the discussion Lavergne reported three cases of retinal detachment in glaucomatous eyes following the topical use of DFP. (4 figures, 5 references)

Peter C. Kronfeld.

Fenton, Robert H. **The incidence of retrolental fibroplasia in Switzerland.** Ophthalmologica **133**:1-11, Jan., 1957.

From the number of reported cases and from a small survey the author concludes that retrolental fibroplasia is very rare in Switzerland. He attributes this low incidence to the relatively high mortality rate of prematures and the very cautious use of oxygen in Switzerland. There also is the possibility of a few cases having been missed through lack of ophthalmological control in some premature stations. (39 references) Peter C. Kronfeld.

Habegger, H. and Ingalls, T. H. **The retinopathy of immaturity.** Ophthalmologica **133**:288-294, April-May, 1957.

In the eye of the mouse which at the time of birth is still very immature, hyperoxia and hypoxia produce the same retinal changes, viz., edema, hemorrhages, tissue proliferation into the vitreous, and finally atrophy. It should, therefore, not be surprising that retrolental fibroplasia occasionally develops in a newborn that has never been exposed to supplemental oxygen. The retinopathy of immaturity may be interpreted as "a vascular disorder resultant from a disturbance of oxygenation at the very critical moment of differentiation of the retinal vessels." (3 figures, 8 references) Peter C. Kronfeld.

Hartmann, E. and Massin, M. **Retinal manifestations of acute disseminated lupus erythematosis (Libman-Sacks disease).** Ann. d'ocul. **189**:973-987, Dec., 1956.

The authors report two cases of acute disseminated lupus occurring in young women. In the first case the usual diagnostic features were equivocal and the retinal findings led to the proper diagnosis. These consisted of a small number of sharply defined, white exudates near the disc, a slight blurring of each disc and a small hemorrhage at the inferior pole of one exudate. Finding lupus erythematosis cells in the blood confirmed the diagnosis. In the second case the diagnosis had been established before ophthalmoscopy but fundus examination again showed the typical exudates at the posterior pole. The authors discuss disseminated lupus erythematosis at length. (6 fiures, 11 references) David Shoch.

Huber, O., Franceschetti, A. and Dieterle, P. **The differential diagnosis between fundus albipunctatus with hemeralopia and Oguchi's disease.** Ophthalmologica **133**:283-287, April-May, 1957.

Fundus albipunctatus with hemeralopia is a rare nonprogressive congenital anomaly which may be difficult to distinguish

from Oguchi's disease. A pertinent case in a 60-year-old man is reported. Aside from a marked deuteranomaly all photopic functions are within normal limits. Discs and retinal vessels are normal. The retina at the posterior pole is slightly gray. Innumerable yellowish-white, round or comma-shaped dots are located deep within the retina in circinate fashion. Dark-adaptation tests show delayed primary and completely absent secondary, that is rod adaptation. The electroretinogram is normal, in sharp contrast to Oguchi's disease in which the a-wave is exaggerated and the b-wave entirely absent. (3 figures, 17 references)

Peter C. Kronfeld.

Lopez Marin, Ignacio. **The diastolic arterial retinal pressure in pregnancy.** Arch. Soc. oftal. hispano-am. 17:231-238, March, 1957.

The general blood pressure and the retinal diastolic arterial pressure were measured in 84 normal pregnant women. The ocular tension was measured on the following day, in order to escape errors caused by the effect of the dynamometric manipulations on the intraocular pressure. Whenever it was possible the tests were repeated three days after delivery. The diastolic arterial retinal tension was measured with the Bailliart ophthalmodynamometer. A ratio between the two diastolic pressures was determined. The conclusions are that the retinal diastolic pressure in pregnancy is neither elevated as reported by Abillart nor diminished as found by Vancea. There was also no significant difference in the data obtained before and several days after delivery. The retino-humeral coefficient at the end of pregnancy was 0.62, and several days after delivery 0.58; this difference of 0.4 is insignificant, and may be due to the variation in intraocular pressure in pregnancy. (3 tables)

Ray K. Daily.

Miratynska-Rusinowa, Elzbieta. **Classification of eyeground changes in diabetics.** Klinika Oczna 27:125-132, 1957.

The author describes changes in the eyegrounds which occur in various stages of diabetes; the pathogenesis of each change is discussed. He feels that the term retinitis is improper and that names for stages, such as proliferative, hemorrhagic or diabetic, are incorrect. Because the blood vessels of the retina are the main tissue involved, the following classification is suggested: 1. angiopathia retinae diabetica, characterized by the presence of venous congestion, microaneurisms, small hemorrhages and exudates without damage to the retina; 2. retinopathia diabetica angiogenes in which vascular changes have produced white exudates and damage to the retina itself; and 3. angioretinopathia proliferativa diabetica. Proliferation of blood vessels and formation of organized foci in the retina reflect the toxicity of this stage of diabetes. Arteriosclerotic changes may exist at the same time and aggravate the condition of the eye. It is frequently hard to distinguish diabetic changes from arteriosclerotic ones. (11 figures, 19 references)

Sylvan Brandon.

Mylius, K. and Witt, G. **The treatment of central vein thrombosis with thrombocid.** Klin. Monatsbl. f. Augenh. 131:145-164, 1957.

75 patients were treated with this heparine derivative during the last six years at a hospital in Hamburg. Better results were usually obtained with a higher dosage of the drug. Only seven patients experienced a loss of vision during the treatment. Out of 47 eyes which could be followed, 12 lost some vision after the treatment. Only a single patient suffered from bleedings (nose). (19 figures, 2 tables, 87 references)

Frederick C. Blodi.

Paufique, L. and Royer, J. **The diagnostic value of dysoric nodules in the retina. Their importance in the diagnosis of collagen diseases.** J. de méd de Lyon 899:583-587, June 20, 1957.

The nodules referred to are cotton wool patches and the histologic substrate is the cytoid body. They are distinguishable ophthalmoscopically and have great diagnostic value. In the absence of signs of hypertensive vascular disease they suggest a collagen disease and they also occur in severe hepatic diseases. (3 figures, 20 references) F. H. Haessler.

Rintelen, F., Hotz, G. and Wagner, P. **Clinical and experimental observations concerning the toxic effects of piperidinephenothiazine upon the pigment epithelium.** Ophthalmologica 133:277-283, April-May, 1957.

The characteristic retinal disease due to NP207 Sandoz was observed in 24 inpatients of the Psychiatric Clinic of the University of Basle. The sensitivity of the retina to the drug varied considerably. Most of the affected patients had received 400-800 mg. per day for two or more months. In a few patients a daily dose of 800 mg. for just a few days produced the typical disease. Discontinuation of the medication was followed in most cases by an appreciable improvement in vision, although the pigmentary changes in the eyeground seemed to progress for some time. Reexamination of 15 cases a year later revealed considerable improvement from the standpoint of function as well as of ophthalmoscopic findings. Animal experiments with large doses of NP207 turned out negative, both ophthalmoscopically and histologically.

In the light of these clinical and experimental findings, NP207 appears to act principally on the pigment epithelium and not on the visual cells. The drug probably interferes with the formation and regeneration of visual purple. At the same time the pigment cells are stimulated to invade the retina proper where they, apparently, cause no appreciable damage. The effects of NP207 have very little in common with the primary or secondary pigmentary degenerations of the human eye. Peter C. Kronfeld.

Sorsby, Arnold. **Some dominantly inherited central fundus lesions.** Acta Genet. & Stat. 7:292-295, 1957.

Sorsby calls attention to seven ocular affections which are now known to be transmitted in a dominant pattern of inheritance and are characterized by a central lesion in the fundus. Two of them are congenital and five are abiotrophies. The congenital defects are congenital macular coloboma and macular cysts. The abiotrophic disturbances are central areolar choroidal sclerosis, macular dystrophy, Doyne's dystrophy, generalized choroidal sclerosis and generalized fundus dystrophy.

Differential diagnosis of these disturbances is of some importance because of the prognosis. The two congenital lesions are easily and reliably recognized. Central areolar choroidal sclerosis, macular dystrophy and generalized choroidal sclerosis are indistinguishable in the early stage but soon develop characteristic patterns. Macular dystrophy must, however, be distinguished from "senile" macular degeneration. Doyne's dystrophy has a characteristic pattern of which the colloid bodies on the nasal margin of the disc are an important element. The prognosis is good for Doyne's dystrophy and fairly good for central areolar choroidal sclerosis, macular dystrophy and macular cyst. It is poorest for generalized fundus dystrophy. (11 references) F. H. Haessler.

Stark, Hilmar. **The effect of heparine on atherosclerotic macular degenerations.** Klin. Monatsbl. f. Augenh. 131:165-178, 1957.

The clearing effect of heparine on certain lipoproteins is here supposed to be the therapeutic principle. The macular degeneration goes first through a phase of exudation. This may be a disadvantage and can be alleviated with cortisone. The dosage is usually small. Contraindications are: advanced age and severe systemic diseases. In early degenerations 12,500 units are given daily for 20 injections. In more advanced degenerations 5,000 units are given every second day for two weeks. No details of results and no controls are described. (7 figures, 22 references) Frederick C. Blodi.

12

OPTIC NERVE AND CHIASM

Blagojevic, M. and Abrambosic, M. **Lesions of the optic nerve during tuberculous meningitis.** Bull. et mém Soc. franç. d'opht. **69**:315-327, May, 1956.

During 1954 and 1955, 250 patients with tuberculous meningitis were regularly examined ophthalmoscopically. Changes of the optic nerve either in the form of a progressive descending optic atrophy or papilledema of varying degree were seen more frequently than miliary tubercles of the choroid. Lesions of the optic nerve and their sequelae therefore were considered to be more significant than hematogenous dissemination of the tuberculous disease. The cause of the optic atrophy was ascribed to interstitial tuberculous neuritis, opticochiasmatic meningeal adhesions and occasionally to tuberculoma of the chiasm. The atrophy was most often sector shaped. Mild discoloration of the disc was more frequent than chalky white pallor. The edema of the disc was ascribed to inflammatory changes of the chiasma and the optic nerves, to the presence of an inflammatory hydrocephalus and edema of the brain and to neurocirculatory disturbances of the centers in the diencephalon. Pathologic specimens showed interstitial perineuritis with collateral edema of the nerve fibres and more or less pronounced inflammatory types of hydrocephalus and edema of the brain. Variable defects in the visual fields proved the presence of both mechanical and inflammatory factors in the etiology of papilledema. Concentric contractions of the fields with and without central scotoma were found as often as central scotomas with abnormalities of the peripheral fields. In spite of the fact that descending optic atrophy was observed mostly in fatal cases, the appearance of the optic disc was not evaluated as a prognostic sign in the individual case. A statistical review of the pertinent facts concerning general and ocular complications of tuberculous meningitis is included. (24 references)

Alice R. Deutsch.

Cartasegna, F. **Periston "N" in the treatment of tobacco-alcohol retrobulbar neuritis.** Riv. oto-neuro-oftal. **32**:267-278, May-June, 1957.

Periston "N" is a slightly viscid liquid consisting of polivinylpyrrolidon together with the chlorides of sodium, calcium, potassium, and magnesium, and a small amount of sodium bicarbonate. This preparation was given intravenously to 22 patients with tobacco-alcohol retrobulbar neuritis. The most gratifying results were obtained in young patients and in those who have had the neuritis for only a short time. The mechanism of action of this agent is apparently through the removal of the toxic catabolites from the blood and the tissues. (11 references)

William C. Caccamise.

Chamlin, Max. **Visual field defects due to optic nerve compression by mass lesions.** A.M.A. Arch. Ophth. **58**:37-58, July, 1957.

In a study of patients with sphenoid ridge meningioma, olfactory groove

meningioma, orbital tumor, and frontal meningioma, the author points out that a "truly discrete central scotoma" is an uncommon field defect. In the author's experience a mass lesion in front of the chiasm most often causes a peripheral defect. Central loss usually results from advancement of the peripheral defect. Early peripheral defects which precede central ones go unnoticed by the patients, hence, are seldom discovered clinically. (5 figures, 1 table, 19 references)

G. S. Tyner.

Doggart, J. H. **Infantile optic atrophy.** Tr. Ophth. Soc. U. Kingdom. 76:127-135, 1956.

The author recommends the use of an anesthetic for examination of the ocular fundus in infants. Disc pallor, the cardinal sign of optic atrophy, is common to all types. Primary optic atrophy (applied especially to degenerative cases) has clear-cut disc margins, deepening and perhaps enlargement of the physiologic cup, and occasionally slight recession of the whole disc and deterioration of the retinal vessels. Secondary optic atrophy, a sequel of disc edema, is characterised by pallor, blurring of margins and obliteration of the physiologic cup.

The variation in color makes color inadequate as a quantitative index of atrophy. An infant's disc is paler than the disc of children and adults and this may be due to delayed myelination. The symptoms that might suggest optic atrophy are the "blank stare," and other factors such as spasticity, deafness and mental backwardness. Temporizing in the prognosis is desirable in most infants.

Beulah Cushman.

François, P., Woilliez, M. and Asseman, R. **The window scotoma in certain diseases of the optic nerve.** Bull. et mém. Soc. franç. d'opht. 69:279-288, May, 1956.

The interpretation of central scotomas often has been quite difficult because of certain erroneous conceptions in the exact structure and arrangement of the macular bundle. Attempts were made to dissociate these defects, to separate them by their minimal dissimilarities and to assign them to particular groups, namely the perimacular central scotoma, the fenestrated scotoma and the typical central macular scotoma. Fenestrated scotomas were observed in four patients whose history is reviewed in detail. In all four patients the visual disturbance started suddenly with a large, dense, central scotoma and concentric contraction of the visual fields. After a certain length of time an improvement of vision occurred. In a central area of about one degree the patients could see again. The small area of visual perception could be sharply outlined in an area of otherwise dense scotoma. The character of the scotoma was suggestive of a disease of the optic nerve, and not of a disease of either the retina or the choroid; because of the recuperation of just the foveolar fibers the lesion was referred to the posterior orbital part of the optic nerve. At this place the foveolar and macular bundle are located in the center of the nerve and get their blood supply through the central branches of the central artery. The most central bundle, corresponding to the foveolar fibers, has the most adequate blood supply. This fact may be the reason for the recovery of these special fibers. The cause of the optic nerve disease in the four patients discussed is ascribed to various factors, multiple sclerosis in case 1, tuberculous meningoencephalitis in case 2, Leber's disease in case 3 and a mine explosion in case 4. The use of vasodilating drugs was suggested in preference to neurosurgical interventions because of the danger of further disturbing the delicate nutrition of the foveolar fibers by additional rupture of capillaries and arteries, accidents which cannot be

avoided in operative procedures of this kind. (6 figures, 5 references)

Alice R. Deutsch.

Friedman, M. Wallace. **Bilateral papilledema in otherwise well patients.** A.M.A. Arch. Ophth. **58**:59-65, July, 1957.

A patient with bilateral papilledema showing no other physical abnormality than obesity is reported. The papilledema was self-limited and resulted in a favorable visual result. (2 figures, 21 references) G. S. Tyner.

13

NEURO-OPHTHALMOLOGY

François, J., Hoffmann, G., vander Eecken, H. and Verriest, G. **Intracranial cholesteatomas.** Ophthalmologica **133**: 369-383, June, 1957.

Two cases of cholesteatoma, one in the cerebello-pontine and one in the suprasellar region, are reported in detail. (12 figures, 17 references)

Peter C. Kronfeld.

Kuellenberg, Ernst. **A case of syringomyelia and syringobulbia with neuroparalytic keratitis.** Ophthalmologica **133**: 269-271, April-May, 1957.

Severe but apparently nonprogressive disturbances of sensibility in the head and neck area and trophic changes of the hands were the outstanding symptoms in the case reported. In accordance with the concept of status dysraphicus the spinal cord lesions are attributed to faulty closure of the central canal during intrauterine development.

Peter C. Kronfeld.

Law, Frank W. **Migraine.** Tr. Ophth. Soc. U. Kingdom, **76**:153-165, 1956.

In the study of 100 consecutive patients, aged 11 to 70 years, the author concludes that the correction of the ocular abnormality contributed toward the relief of symptoms in the majority. The use of ergot preparations and hyperoxygenation in the early stages has been of definite help in some patients who did not respond to correction of the ocular abnormalities. (1 figure, 2 tables)

Beulah Cushman.

Spadavecchia, V. **An additional contribution to the recognition of nasociliary neuritis: true (interstitial) neuritis, and degenerative (parenchymatous) neuritis.** Riv. oto-neuro-oftal. **32**:325-347, May-June, 1957.

In this fourth article concerning nasociliary neuritis the author discusses the diagnosis, the prognosis, and the therapy of this condition.

William C. Caccamise.

Vanderfield, Geoffrey. **Periodic headache associated with cerebral aneurysm, with report of a case successfully treated by surgery.** M. J. Australia **2**:289-290, 1957.

The patient had attacks simulating migraine but developed right ophthalmoplegia. Cerebral angiography showed a large saccular aneurysm arising from the distal part of the internal carotid artery. Relief of symptoms occurred after direct surgical attack upon the aneurysm by placing a silver clip across its neck. (1 figure, 3 references) Ronald Lowe.

14

EYEBALL, ORBIT, SINUSES

Cory, J. W. E. **Unilateral exophthalmos resulting from sclerosis of the sphenoid bone.** Brit. J. Ophth. **41**:434-437, July, 1957.

A 53-year-old woman had a moderate degree of unilateral exophthalmos. The only pathologic findings were X-ray evidence of sclerosis of both wings of the sphenoid bone extending to the zygoma and frontal bones. The lesion progressed

very slowly and no treatment was recommended. (5 figures, 7 references)

Morris Kaplan.

Lederman, M. **Technique of radiation treatment of orbital tumours.** Brit. J. Radiol. **20**:469-476, Sept., 1957.

The procedures of radiation in treatment of orbital tumors are described in elaborate detail. (11 figures, 1 table, 3 references)

Irwin E. Gaynon.

Palich-Szanto, O. **Differential diagnosis of orbital diseases.** Klin. Monatsbl. f. Augenh. **131**:260-266, 1957.

Three cases are reported. One woman had a mucocele, another old woman had an orbital periostitis, and a young man had a dermoid cyst. A close cooperation with the ear-nose-throat department is necessary to arrive at a correct diagnosis.

Frederick C. Blodi.

Rose, D. and Taylor, C. **Multiple myelomatosis affecting the orbit.** Brit. J. Ophth. **41**:438-439, July, 1957.

A 56-year-old woman noted a rather sudden, severe and progressive left-sided exophthalmos. Radiographic examination revealed widespread multiple myelomata with erosion of the roof of the orbit and the presence of a tumor invading the orbit. Necropsy findings revealed the large tumor, attached to the dura, which had eroded the supraorbital plate and passed downward into the orbital space. (1 figure, 1 reference)

Morris Kaplan.

Van Buren, J. M., Poppen, J. L. and Horrax, G. **Unilateral exophthalmos; consideration of symptom pathogenesis,** Brain **80**:139-175, 1957.

Many studies of expanding lesions of the orbit based upon a large series of cases have concerned themselves primarily with the frequency of occurrence and the histologic features of the neoplasms. It has, however, occurred to us that if these lesions be considered as experimental intrusions upon the orbit, features of fundamental interest might be adduced from their study. A series of 86 cases of unilateral exophthalmos seen and treated on a neurosurgical service were reviewed for consideration of the pathological and clinical characteristics of lesions producing this symptom.

On the pathological side, the invasiveness of meningiomas becomes evident once they pass into mesenchymal tissues, although their rate of growth is slow. Partial removal of gliomas of the optic nerve and chiasm may be attended by remarkably prolonged periods without recurrence of increase of symptoms. The frequency of fibrous dysplasia in the present series was unexpected and suggests that the lesion may at times go unrecognized. The term cholesteatoma proved confusing in the present material and it appeared that the lesions could be reclassified with advantage under more specific terms. Of eight cases presenting unilateral exophthalmos without tumour, two were tentatively considered as examples of thyrotropic exophthalmos while six were associated with hypertension and two of these with glaucoma. Further cases are required to establish whether this indeed forms a syndrome and what part, if any these features play in the pathogenesis of exophthalmos.

On the clinical side, macular, peripheral and intermediate defects in the visual fields were found and no good evidence was disclosed to explain the variations. Venous obstruction in the posterior orbit appears to be an important factor in the production of exophthalmos and the inadequacy of the anterior venous drainage from the orbit is noted. Papilledema was seen with lesions posteriorly in the orbit, but not anteriorly, and seemed satisfactorily explained on a venous congestive basis. The relatively greater sensitivity of the parasympathetic fibres to pressure as

compared with the motor nerve fibres has been demonstrated and indirect evidence suggests that sympathetic overaction has little to do with the mydriasis in orbital lesions. Review of the extraocular muscle palsies suggests that direct pressure upon the muscle is a more frequent cause of inactivation than is compression of its nerve supply in the orbit. The almost selective vulnerability of the superior rectus muscle to pressure was a frequent finding. Authors' summary.

Yasargil, M. G. **Angiography in the differential diagnosis of unilateral exophthalmos.** Ophthalmologica 133:212-214, April-May, 1957.

In 68 out of 110 cases of unilateral exophthalmos angiography through the common carotid was considered indicated and actually performed. With the new contrast material urographin (in 60 percent concentration) the author succeeded in filling the ophthalmic artery and its branches in 87 percent of the cases. Phlebography of the orbital region proved feasible through injection of the angular vein. No complications are reported though the author warns against carotid angiography in acute cerebral vascular accidents or head traumas.

Peter C. Kronfeld.

15
EYELIDS, LACRIMAL APPARATUS

Ayberk, N. F. **Primary sarcoma of the four lids, treated with nitrogen mustard.** Bull. et mém. Soc. franç. d'opht. 69:255-262, May, 1956.

Chemotherapy alone or in combination with irradiation has been rarely used in malignant tumors of the eyeball and its adnexa. The history of a 70-year-old man is reported. He suddenly developed swelling and induration of all four lids. His general health was good with the exception of a mild anemia of long standing. He also showed swelling of the regional lymph nodes and pronounced mediastinal adenopathy. Biopsy verified the clinical diagnosis of lymphoblastic sarcoma. Chemotherapy was started and consisted of 7 mg. Dichlorene on six successive days. Six weeks later a similar dose, namely 7 mg. Dichlorene was given on five successive days. A practically complete regression of the lid tumors and of the glandular metastasis took place. After a four-year observation time the patient was found to be well. This excellent therapeutic result was even more remarkable since the occurrence of multiple sarcomas of the lids is considered to be a deadly disease. (5 figures, 6 references) Alice R. Deutsch.

Calmettes, L., Deodati, F., Planel, H. and Bec, P. **The influence of hormones on the lacrimal gland (experimental results in the guinea pig).** Bull. et mém. Soc. franç. d'opht. 69:263-270, May, 1956.

The pathogenesis of Gougerat-Sjøgren's syndrome has been the subject of many papers; the cause of this disease was first ascribed to multiple vitamin deficiency, later to diffuse endocrine disturbances. The endocrine etiology was sustained by the fact that the syndrome is mostly seen in women of menopausal age or later. Estrone (folliculin), however, decreases the function of the lacrimal gland; episodes of oversecretion of estrone occur just during the menopause and these periods of oversecretion of estrone could precisely contribute to the increasing atrophy of the lacrimal gland. The specific modifications of the lacrimal glands under the influence of different hormones were studied on 26 male and 26 female guinea pigs. Harder's retrobulbar glands were examined in preference to the lacrimal gland. The structure of the acini of Harder's gland show dimorphism in normal animals; 12 males and 12 females were given testosterone acetate and es-

trone respectively. The acini of the males showed an increase in diameter and those of the females showed hypotrophy. After castration the lacrimal gland of 12 male guinea pigs was reduced in size without change in the structure of the cells, but reduction in the diameter of the acini, and similarly in 12 females the lacrimal gland was slightly heavier and the diameter of the acini had increased. These experiments are in accordance with various other experiments and are a valuable contribution towards an understanding of the physiopathology of the lacrimal gland. (4 figures, 3 references)

Alice R. Deutsch.

Cordier, J. and Algan, B. **Localization of Besnier-Boek-Schaumann disease in the lacrimal gland.** Bull. et mém. Soc. franç. d'opht. 69:270-277, May, 1956.

A solitary localization of sarcoidosis in the lacrimal gland is rare indeed and two observations are reported. One occurred in a 49-year-old man, in excellent general health, but with a painless enlargement of the right lacrimal gland. The routine intra-dermal tuberculine test was negative; the B.C.G. test was positive, proving a mild tuberculous allergy. The other patient, a 57-year-old woman had bilateral lacrimal adenopathy. Physical examination, divers laboratory tests, X-ray films, intracutaneous tuberculin and B.C.G. test were negative. The biopsy specimen of both patients showed diffuse lymphoid infiltration and follicle formation, giant cells surrounded by epithelioid cells and lymphocytes but no caseation. The pathologic diagnosis was tuberculosis or sarcoidosis. It was emphasized that neither the clinician nor the pathologist alone could possibly make the diagnosis in an isolated lesion and that the close collaboration of both is neccessary. The positive B.C.G. test in the first case was interpreted as a turning point in the tuberculin sensitivity, either towards early recovery or towards an outbreak of tuberculosis. (3 figures, 5 references)

Alice R. Deutsch.

Kharchenko, A. **A method of prevention of recurrence of obstruction of the lacrimal ducts after the operation of anastomosis.** Vestnik oftal. 4:51-55, July-Aug., 1957.

During the last eight years, 315 dacryocystorhinostomies were done in 298 patients, for uncomplicated dacryocystitis in 202, for bilateral lesions in 20, and for complicated, phlegmonous dacryocystitis in 59 patients. Favorable results (patency of the duct) were obtained in 313 cases (99.4 percent). This was confirmed by the fluorescein test.

The technique of the operation is described in detail. The last step is the one that gives the good functional result: after the anterior sutures are applied, a gauze tampon soaked in sterile oil and sprinkled with sulpha powder is put through the nose into the anastomosis. The cavity is filled with the tampon, after which the catgut sutures are tied over the tampon.

The advantages of this procedure are: the tamponade prevents hemorrhage, it works as drainage in the first hours after the operation, but chiefly prevents the apposition and gluing of the cut anterior and posterior surfaces of the edges of the mucous membrane. The tampon, by straightening the cavity of the anastomosis, acts favorably in the healing of the wound surfaces. The sulpha powder prevents infection. The oiled tampon stays moist, does not press on the mucous membrane (as a gold or rubber tube does) and on removal does not traumatize the newly formed epithelium. Since the tampon is placed in a natural cavity (the sac and the nose), it does not act as a foreign body and does not cause a reactive inflammation. The tampon is removed easily in 48 hours after the operation; no

irrigation of the lacrimal duct is necessary. The epithelization of the mucous membrane at the site of the incision takes place in about two days.

Olga Sitchevska.

Kozakiewicz, A., and Mielnik, I. **Plastic reconstruction of the lids and the orbital cavity with the help of Filatov's round flaps.** Klinika Oczna **27**:121-124, 1957.

A case of plastic reconstruction of the eye socket and the lids is presented. A man, 25 years of age, had crushing injury to the left orbit which destroyed the lids and the eyeball. Extensive surgery had already been performed, including Filatov's round flap with the base in the orbital area, before the authors started their work. The flap was split lengthwise horizontally, the subcutaneous fat was removed and the skin was turned and fashioned to line the bony orbit and to form both lids. A prosthesis was put in immediately to facilitate shaping of the socket. The eyebrow was formed by transplanting a piece of skin with hair from the back of the neck. (4 figures, 3 references) Sylvan Brandon.

Rosado Rodriguez, Enrique. **Dacryocystitis in infancy.** Arch. Soc. oftal. hispano-am. **17**:239-241, March, 1957.

Rosado advocates early probing of obstructed lacrimal passages in infants, pointing out that the older the infant the more difficult it is to restrain him, and that after six months the infected mucous membrane of the lacrimal sac loses its elasticity and probing becomes ineffective. Ray K. Daily.

16
TUMORS

Binkhorst, C. D., Schierbeek, P. and Petten, G. J. W. **Neoplasms of the notochord. Report of a case of basilar chordoma with nasal and bilateral orbital involvement.** Ophthalmologica **133**:12-22, Jan., 1957.

The report concerns a case of an intracranial neoplasm affecting originally the optic chiasm and invading both sphenoids. The initial subjective symptoms were headaches and loss of vision. The neoplasm slowly destroyed the sphenoids and invaded both orbits and the nasal cavity. At the age of 70 years the patient was seen by the authors because of nasal obstruction. A glassy polyp-like tissue protruded from one nostril. "In three sessions about 65 grams of tumor tissue were removed by the endonasal route after local application of pontocaine and epinephrine." The histologic diagnosis was chordoma. (8 figures, 19 references)

Peter C. Kronfeld.

Centanni, L. and Morello, F. **Ocular changes in some tumors of the sellar and juxtasellar region and their modifications after radiotherapy.** Riv. oto-neuro-oftal. **32**:279-324, May-June, 1957.

The authors present their impressions based on the study of 50 cases of neoplasm of the sellar and juxtasellar region. The correlation of X-ray therapy with changes in the eye symptoms was evaluated. (10 figures, 5 tables, 34 references) William C. Caccamise.

Dunphy, Edwin B. **The role of radioactive phosphorus in the diagnosis of ocular malignancy.** Tr. Ophth. Soc. U. Kingdom **76**:137-152, 1956.

The author and his colleagues use radioactive phosphorus (P^{32}) in the detection of intraocular tumors. In several papers already published it had been definitely shown by radio assay that malignant melanomas concentrate P^{32} to a greater degree than other ocular tissues. A variation between the pathologic and the normal eye of at least 30 percent is necessary to justify a presumptive diagnosis of neoplasm. In 1952 Trevor-Roper,

Newton and Nicholson in England reported disappointing results in two of eight cases of proven malignancy.

The author reports on the use of P^{32} in a series of 58 cases. The method of performing the tests consisted of giving 750 microcuries of P^{32} intravenously as a buffered sodium phosphate to an adult of average size. In small, frail adults only 500 microcuries were given and much less in children. Approximately 24 hours later the counting is begun with the patient in the prone position. After use of two or three drops of ½-percent pontocaine solution, the specially designed Geiger counter is placed as close as possible to the suspected lesion for one minute and the counts recorded. An average of three counts is taken from each location.

The author emphasizes the fact that the use of P^{32} should be regarded as a diagnostic aid and not as a positive diagnostic test. (6 figures, 2 tables, 8 references) Beulah Cushman.

17

INJURIES

Funder, Wolfgang. **Unusual course of a siderosis.** Klin. Monatsbl. f. Augenh. 131:258-259, 1957.

A 52-year-old man had an intraocular foreign body for 19 years. The eye became finally blind and painful. The iris had been brown for 10 years but became again blue. This part of the siderosis is therefore reversible and may be due to a fibrotic capsule. (13 references)

Frederick C. Blodi.

Gilsenan, L. D. **A fatal case of parathion poisoning.** M. J. Australia 2:251, 1957.

The effects of organic phosphate insecticide poisoning are due to intense anticholinesterase activity. Ocular manifestations are blurred vision, constricted pupils and twitchings of the eyelids. A fatal case is described. Ronald Lowe.

Guillot, P. and Farharier, G. **Extraction of intraocular magnetic foreign bodies.** Ann. d'ocul. 189:880-883, Oct., 1956.

The authors' innovations are: use of curare in addition to local anesthesia; paracentesis of anterior chamber to diminish chance of vitreous loss; and use of the electric cautery to perforate the choroid. The approach is always via the posterior route. (3 figures) David Shoch.

Milovidova, A. **Burns of the eye and their treatment.** Vestnik oftal. 4:45-50, July-Aug., 1957.

This is a review of the treatment of burns of the eye in 430 patients hospitalized at the Novosibirsk Medical Institute during 10 years (1945 to 1954). Of these, 201 patients were injured with lime and alkali burns; 69 with acids, 111 had thermal burns and 49 had burns of miscellaneous origin. Burns of the first degree were observed in 210 patients, of the second degree in 176 and of the third degree in 94 patients. The author classified the second and third degree burns as those in which the conjunctiva is necrotic; in the third degree burn the cornea is involved, it is opaque and insensitive, with nearly complete loss of vision. The gravity of alkali burns of the eye is stressed, also the urgency of immediate care.

Milovidova used autohemotherapy (instead of Denig's transplantation of the mucous membrane from the lip). 25,000 to 30,000 units of penicillin, mixed with 0.3 cc. of the patient's blood were injected subconjunctivally daily or every other day, up to 7 times in severe cases. The healing was without the formation of symblepharon, ankyloblepharon or conjunctival bands. By this new method of treating eye burns, the vision was restored in nearly 90 percent of the patients. The only cases in which the eye responded poorly were burns of third degree with ammonia. (3 tables)

Olga Sitchevska.

18

SYSTEMIC DISEASE AND PARASITES

Bonamour and Bouysset. **Liver and eye.** J. de méd. de Lyon **899**:589-594, June 20, 1957.

The authors summarize a number of diseases of the liver in which ocular disturbances occur. They point out that there is no ocular disturbance which is characteristic of liver ailment and might be called "the hepatic eye," yet abnormalities in the eye can be the diagnostic sign which leads to recognition of a hepatic lesion, for example, miliary tuberculosis. The ophthalmoscopic manifestation of a dysoric disturbance is often correlated with the evolution of a liver deficiency and hence may be of great prognostic value.

F. H. Haessler.

Brand, I. **Changes of the intraocular pressure in Parkinsonism.** Ophthalmologica **133**:53-60, Jan., 1957.

The eye findings in 23 cases of postencephalitic, idiopathic or arteriosclerotic Parkinsonism are reported. Ocular hypotony seems to be a characteristic symptom of the postencephalitic form of Parkinsonism. This hypotony is attributed to a central (diencephalic) degenerative process. (36 references)

Peter C. Kronfeld.

Cristini, G., Tolomelli, E., Schiavi, L. and Rumpianesi, G. **The thrombocytes in cases of diabetic retinopathy.** Ophthalmologica **133**:119-130, Feb., 1957.

This cooperative study by internists and ophthalmologists revealed the presence, in the thrombocytes of diabetics with retinopathy, of polysaccharides which differ considerably in their histiochemical behavior from those found in the thrombocytes of normal individuals. The histiochemical abnormalities of the thrombocytes of diabetics did, however, not differ appreciably from those found in other diseases affecting primarily the blood platelets. The authors are inclined to attribute to the platelet changes demonstrated by them a significant role in the pathogenesis of diabetic retinopathy. (5 tables, 15 references)

Peter C. Kronfeld.

Goerger, F. **A case of intraocular cysticercosis.** Ann. d'ocul. **189**:988-992, Dec., 1956.

The author reports the case of a young man with a history of Taenia solium infestation. He complained repeatedly of blurred vision and eventually developed an inferior retinal detachment. Finally a contractile cyst was seen in the vitreous and removed via a sclerochoroidal incision. A second operative procedure was performed to reattach the retina with diathermy. One month postoperatively three-fourths of the retina was seen to be reattached. (1 figure)

David Shoch.

Juzwa, Jozefa. **Eye symptoms in osteomyelitis of the maxilla in infants.** Klinika Oczna **27**:163-168, 1957.

The author feels that osteomyelitis of the maxilla is relatively frequent and not always recognized. The general condition of the patient is always very serious and symptoms are mainly of a septic character. Ocular symptoms frequently resemble purulent dacryocystitis or orbital cellulitis. Examination of the mouth may disclose the presence of purulent fistula. Four cases in infants below one year of age are described; streptococcus was found in three and no organisms in one. Intensive treatment with antibiotics saved the children's lives. (3 references)

Sylvan Brandon.

Wilson, M. M. **Human brucellosis in Victoria.** M. J. Australia **2**:413-417, 1957.

Human brucellosis has been a notifiable disease since 1937. The author reviews 121 probable cases diagnosed by the Pub-

lic Health Laboratory between 1954 and 1956. Seven patients had uveitis.

Ronald Lowe.

Wybar, Kenneth. **An assessment of modern views on the nature of endocrine exophthalmos.** Tr. Ophth. Soc. U. Kingdom 76:107-125, 1956.

As no true understanding of the basic nature of the exophthalmic process has been evolved, the author uses the accepted chemical regulation and coordination of bodily functions as an impetus for the study of experimental exophthalmos. Two main forms of endocrine exophthalmos are accepted, one associated with thyrotoxicosis and the other thyrotropic.

The exophthalmos of toxic diffuse goiter and the progressive exophthalmos usually follows antithyroid treatment. Thyrotoxic exophthalmos as a primary manifestation of Graves' disease is to a large extent an apparent exophthalmos due to lid retraction, and the lid retraction appears to be an independent phenomenon.

The thyrotropic or progressive form of exophthalmos is discussed under various headings: increase in water content, mucin content, fibrosis and lymphocytes. Because of the relation of adrenocorticotropic hormone (ACTH) and adrenocortical hormone (cortisone) to exophthalmos it seems reasonable to advocate the use of these hormones in cases of exophthalmos, particularly in the early stages before the orbital changes become established and irreversible. (4 figures, 106 references)

Beulah Cushman.

19

CONGENITAL DEFORMITIES, HEREDITY

Badtke, G. **Nature and genesis of so-called atypical colobomas of the inner coats of the eye and of the optic nerve.** Klin. Monatsbl. f. Augenh. 131:1-18, 1957.

An extensive review of the literature is given and a number of new cases are reported. Most interesting are combinations of an atypical coloboma of the iris with an atypical coloboma of the choroid and the optic nerve. These cases speak against the importance of the physiologic dimples on the optic cup in the etiology of atypical colobomas. Other cases are described in which a typical coloboma in one eye is associated with an atypical coloboma in the other eye. In addition, Sato produced experimentally atypical fetal clefts in amphibian embryos. It is therefore assumed that atypical colobomas are caused by an atypical or accessory fetal cleft and the term "coloboma in an atypical direction" would be more appropriate. (10 figures, 20 references)

Frederick C. Blodi.

François, J. and DeVos, E. **Heredity of disc-shaped cataract.** Acta Genet. & Stat. 7:266-273, 1957.

The authors describe and discuss the appearance of a disc-shaped annular cataract in three generations of a family in which it was transmitted by autosomal dominance. (2 figures, 23 references)

F. H. Haessler.

Meyer-Schwickerath, G., Grueterich, E. and Weyers, H. **Microphthalmos syndromes.** Klin. Monatsbl. f. Augenh. 131: 18-30, 1957.

This syndrome is characterized by a combination of microphthalmos with anomalies of the face and the extremities. Two types are distinguished. a. The so-called oculo-dento-digital dysplasia is discussed on the basis of two observations and two more cases found in the literature. Microphthalmos may be accompanied by an atrophy of the iris. There is hypoplasia of some of the middle phalanges. The teeth show a yellow color and defects of the enamel. b. Dyscranio-pygophalangia of Ullrich is illustrated by one case. The anomalies are here much more

pronounced and the infants do not survive. (14 figures, 43 references).

Frederick C. Blodi.

Nicetic, B. and Sakic, D. **Nodular degeneration of the cornea associated with color of the iris.** Acta Genet. & Stat. 7:274-276, 1957.

The authors provide the pedigree of four generations of a family in which the corneal lesion and iris color are associated and discuss the theoretical implications of the occurrence of multiple heredity that is the pattern of behavior of several pathologic genes in a family tree. (1 figure)

F. H. Haessler.

Valentin-Gamazo, Ignacio. **Typical, congenital, monocular coloboma of the iris, retina and optic nerve.** Arch. Soc. oftal. hispano-am. 17:165-176, Feb., 1957.

The 15-year-old patient had, in addition to these ocular anomalies, hypertrichosis of the face, a malformation of the palate, and impaired hearing for high and low tones. All these anomalies are of ectodermic origin, caused by an interference in the development of the first branchial arch and cleft. The embryology of the eye during the first four months of gestation and the development of the malformation are described in detail. The association of these anomalies may be considered as the ectodermic syndrome of the first branchial arch. (12 figures, 6 references)

Ray K. Daily.

Williams, R. K. and DuPont Guerry III. **Choroideremia: report of cases and review of literature.** South. Med. J. 50: 1048-1053, Aug., 1957.

Choroideremia is primarily a disease of the choroid having intermediate sex-linked characteristics, showing a progressive form in the male with early nyctalopia leading to blindness and in the female a relatively nonprogressive attenuated form without nyctalopia or defective vision. A brief review of the literature and 11 additional cases, 6 female and 5 male from three generations of the same family, are presented. The value of Aquasol A is mentioned with the utmost reserve, but the authors seem to feel that some of these patients are being helped.

F. H. Haessler.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Dall'Osso, E. **A district ophthalmologist in the 14th century.** Gior. ital. oftal. 9:593-599, Sept.-Oct., 1956.

After an interesting account of ophthalmic news of the fourteenth century, a document is discussed which shows that in the year 1276 a district eye service existed in Perugia. This document, as well as another in the form of a contract between an eye doctor and a patient, dated 28th Sept. 1479, are reproduced. (18 references)

V. Tabone.

Hertzberg, R. **The care of the eyes of the newborn baby.** M. J. Australia 2:402, 1957.

The author reviews the Credé method. It is recommended that with adequate antenatal care and delivery in a modern hospital prophylaxis of ophthalmia neonatorum consist of swabbing the baby's eyes before they are opened. In unsatisfactory surroundings the use of silver nitrate drops is desirable.

Richard Lowe.

Koelbing, M. H. **Felix Platter's contribution to ophthalmology.** Ophthalmologica 133:364-368, April-May, 1957.

Felix Platter (1536-1614) was a Swiss physician who in his "De corporis humani structura et usu . . . libri III" (Basle 1583) gave a remarkably accurate anatomical description of the human eye and in his other writings ("Observationes," "Praxis") made some astute observations regarding various ocular diseases. (4 references)

Peter C. Kronfeld.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Hobart Clemens Johnson, Mankato, Minnesota, died July 23, 1957, aged 60 years.

Dr. Albert Napoleon Lemoine, Sr., Kansas City, Missouri, died August 30, 1957, aged 72 years.

Dr. Clarence Archibald Veasey, Spokane, Washington, died August 11, 1957, aged 88 years.

ANNOUNCEMENTS

MICHIGAN CONFERENCE

The University of Michigan Medical School announces the annual Ophthalmology Conference to be held April 21, 22, and 23, 1958.

The conference will be presented at the University Hospital by Dr. F. Bruce Fralick and staff of the Department of Ophthalmology. Guest speakers will be Dr. Max Chamlin, New York; Dr. Paul A. Chandler, Boston; Dr. S. Rodman Irvine, Los Angeles; Dr. Henry P. Wagener, Rochester, Minnesota; and Dr. George N. Wise, New York.

Applications may be addressed to the:
Department of Postgraduate Medicine
University Hospital
Ann Arbor, Michigan

EAST-CENTRAL MEETING

The eighth annual meeting of the East-Central Section of the Association for Research in Ophthalmology will be held at the Wade Park Manor Hotel, Cleveland Ohio, on Monday, January 6, 1958.

A tentative program of papers to be presented is: "Horner's syndrome," Conrad L. Giles and John W. Henderson, Ann Arbor, Michigan; "Nature and significance of horizontal folds of the retina," J. Reimer Wolter, Ann Arbor, Michigan; "The microcirculation in the bulbar conjunctiva," Edward H. Bloch, Cleveland, Ohio; "Are aqueous humor dynamics influenced by aging? II." W. M. Spurgeon, Cincinnati, Ohio, B. Boles-Carenini and A. Cambiaggi, Universita Cagliari, Italy; "The behavior of the intraocular pressure in the valsalva experiment," David A. Rosen, Kingston, Canada; "The effect of removal of various abdominal organs on the glucose uptake of the lens," Tibor G. Farkas, Cleveland, and John W. Patterson, Vancouver, British Columbia.

"Adenylic acid deaminase in ciliary processes," M. B. Waitzman, and E. J. Ballintine, Cleveland; "Conoid refracting surfaces and conoid lenses," David Volk, Cleveland; "Variability of accommodation during steady fixation at various illuminance levels," Mathew Alpern, Ann Arbor; "Correction for the guessing bias in the Landolt ring test," Jack H. Prince and Glenn A. Fry, Columbus, Ohio; "Retinal and optic nerve atrophy in dogs exposed to Isopropyl oil fog," Jay G. Linn, Jr., Pittsburgh, Pennsylvania; "The electroretinogram in retinitis pigmentosa," Albert D. Ruedemann, Jr., and Werner K. Noell, Detroit, Michigan; "Factors influencing the movement of fluorescein in the cornea," A. J. Beswick, R. Langley, and Clement McCulloch, Toronto, Canada; "Effect of pH, temperature, and ionic environment on the transcorneal potential," Robert W. Modrell and Albert M. Potts, Cleveland; "The transport of tritium labeled water across the isolated cornea," Albert M. Potts and Beatrice Cohen, Cleveland.

FIGHT FOR SIGHT FUNDS AVAILABLE

The Scientific Advisory Committee of the National Council to Combat Blindness, Inc., 41 West 57th Street, New York 19, announces that it is now accepting applications for its 1958-59 fight for sight Grants-in-Aid, Research Fellowships, and Summer (1958) Student Fellowships. In accordance with its program of financing research in ophthalmology and related sciences and encouraging qualified investigators to further research, the Scientific Advisory Committee will consider all applications at its ninth annual meeting scheduled for June, 1958.

The closing date for receipt of completed applications for grants and fellowships has been advanced to March 1, 1958, in order to allow ample time for processing and review. Applications for Summer Student Fellowships will be reviewed in advance of the June meeting. Appropriate forms may be obtained by addressing the Council's new address:

Secretary
National Council to Combat Blindness, Inc.
41 West 57th Street
New York 19, New York

The Scientific Advisory Committee consists of 19 members: 13 ophthalmologists, two physiologists, one biochemist, one microbiologist, one psychologist and one zoologist. The chairman is Charles A. Perera, M.D., associate professor, Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York. The other members are: James H. Allen, M.D., Tulane University; Bernard Becker, M.D., Washington University; Alson E. Braley, M.D., University of Iowa; Frederick Crescitelli, Ph.D., University of California; Arthur G. DeVoe, M.D.,

New York University-Bellevue Medical Center; Dan M. Gordon, M.D., New York Hospital, Cornell Medical Center; Charles Haig, Ph.D., New York Medical College; Michael J. Hogan, M.D., University of California; Peter C. Kronfeld, M.D., University of Illinois; Irving H. Leopold, M.D., Wills Eye Hospital; A. E. Maumenee, M.D., Johns Hopkins University; Karl Meyer, M.D., College of Physicians and Surgeons, Columbia University; Stuart Mudd, M.D., University of Pennsylvania; Lorrin A. Riggs, Ph.D., Brown University; Theodore C. Ruch, Ph.D., University of Washington; Samuel L. Saltzman, M.D., New York Medical College, Flower and Fifth Avenue Hospitals; Kenneth C. Swan, M.D., University of Oregon; Phillips Thygeson, M.D., University of California.

The goal of the National Council to Combat Blindness is the alleviation of defective sight and blindness through the financing of grants and fellowships approved by its Scientific Advisory Committee. The 1957-58 Fight for Sight research allocations amounted to $128,000. These included 24 Grants-in-Aid, 10 Research Fellowships, and 24 Summer Student Fellowships. Awards were made in 16 states (28 institutions) and in Canada, Israel, and Japan.

XVIII International Congress

The XVIII International Congress of Ophthalmology will be held in Brussels, September 8 to 12, 1958, under the presidency of Prof. Leon Coppez. The main subjects chosen by the International Council are:

Orthoptic treatment of strabismus, with Dr. A. Bangerter, St. Gallen, Dr. T. K. Lyle, London, and Prof. J. Malbran, Buenos Aires, as moderators.

Geriatrics in ophthalmology, presented by Prof. M. Buerger, Leipzig, Prof. J. François, Ghent, Prof. G. Jayle, Marseille, and Prof. M. Jayle, Paris.

Symposia on cataract, radioactive isotopes in ophthalmology, electroretinography, and glaucoma will be presented. Sessions will also be reserved for free papers. The International Association for Prevention of Blindness and the International Organization against Trachoma will hold meetings during the congress. French, English, and German are the official languages for the congress.

All correspondence and requests for information should be sent to the general secretary:

Prof. Jules François
15, Place de Smet de Naeyer
Ghent, Belgium

Dallas Meeting

At the sectional meeting of the American College of Surgeons to be held at Dallas, Texas, January 9 through 11, 1958, the following symposia on ophthalmic surgery will be held: "Current trends in corneal surgery," Dr. William B. Clark, New Orleans, and Dr. George P. Landegger, Los Angeles: "Current trends in retinal surgery," Dr. C. Keith Barnes, Fort Worth, Dr. Berton F. Frink, McAllen, and Dr. William L. Spaulding, Fort Sam Houston.

Chicago clinical conference

The Chicago Ophthalmological Society will hold its annual clinical conference on February 21 and 22, 1958, at the Drake Hotel in Chicago.

The guest speakers will include Dr. Wendell L. Hughes, Hempstead, New York, Dr. P. Robb McDonald, Philadelphia, Dr. Phillips Thygeson, San Jose, California, Dr. Lorenz E. Zimmerman, Washington, D.C., and Dr. Eugene R. Folk, Dr. Orville E. Gordon, Dr. William F. Hughes, Dr. Peter C. Kronfeld, Dr. David Shoch, Dr. Derrick Vail, and Dr. Howard L. Wilder, all of Chicago.

In addition to a symposium on the complications of ophthalmic surgery, the subjects will include the zonule of the lens, glaucoma and retinal detachment surgery, plastic surgery techniques, management of glaucoma and cataract, melanotic tumors of the iris, and the structure of the vitreous.

Registration fee for the entire course, including round table luncheons and buffet supper is $45.00, and may be payable to the

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The 14th annual Sanford R. Gifford Memorial Lecture will be delivered by Dr. Phillips Thygeson of San Jose, California, on Friday, February 21, 1958, at the Drake Hotel at 5:15 P.M. All ophthalmologists are invited to attend the Gifford Memorial Lecture and the buffet supper which follows.

Miscellaneous

Schoenberg lecture

The annual Schoenberg Memorial Lecture of the New York Society for Clinical Ophthalmology and the National Society for the Prevention of Blindness was presented on December 2nd at the New York Academy of Medicine, by Dr. Bernard Becker, chief of the Department of Ophthalmology at the Washington University School of Medicine, Saint Louis, whose subject was "Tonography: Experimental and clinical aspects."

Mark J. Schoenberg, M.D., a Rumanian refugee who became a distinguished American ophthalmologist, was a founder of the New York Society and long spearheaded the national society's glaucoma control program. Since his death in 1945, the two societies have joined each year in honoring his memory.

Research in Australia

The Ophthalmic Research Institute of Australia is composed of the following:

Directors: Dr. A. S. Andreson, Melbourne, chairman; Sir Norman Gregg, Sydney, Dr. W. Lochhart Gibson, Brisbane, Dr. A. L. Tostevin, Adelaide, Dr. J. Ringland Anderson, Melbourne, Dr. Arthur Joyce, Melbourne, Prof. Ida Mann, Berth, and Dr. J. Bruce Hamilton, Hobart. The secretary is Dr. W. Deane Butcher, Macquarie

Street, Sidney, New South Wales. On the Research Committee are: Sir Norman Gregg, Prof. Ida Mann, Dr. J. Ringland Anderson, and Dr. J. Bruce Hamilton.

Societies

Kansas City meeting

The program for the ophthalmology session at the November 21st meeting of the Kansas City Society of Ophthalmology and Otolaryngology included: Eye surgery, a case report by Dr. John Bradford; presentation of interesting cases, Dr. Charles Crokett, leader; slides of the anterior chamber by Dr. Travis Robison; and two lectures by the guest speaker, Dr. T. E. Sander, Saint Louis: "Choice of glaucoma surgical procedures based on pathologic specimens," and "Personal experiences in three disease entities."

European Society of Ophthalmology

The European Society of Ophthalmology was founded on May 14, 1957, by delegates from various European countries who met in Paris under the presidency of Dr. M. P. Bailliart.

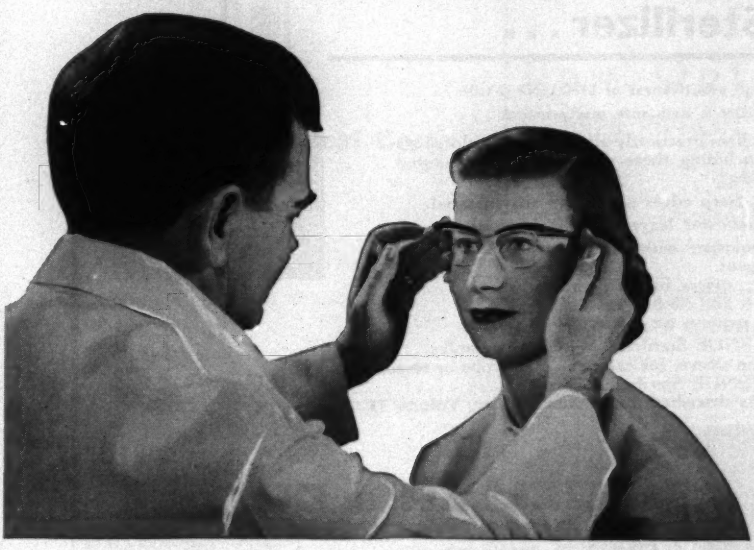
Those present at the meeting were: Prof R. Thiel, Germany; Ladekarl, Denmark; Arruga, Spain; Prof. Vannas, Finland; J. Sedan, France; Keith Lyle, Great Britain; Prof. Charamis, Greece; Prof. Hagedoorn, Holland; Werner, Ireland; Prof. Maggiore, Italy; Prof. Lopez de Andrade, Portugal; Prof. von Bahr, Sweden; Huber, Switzerland; Prof. Vejdovsky, Czechoslovakia; Ayberk, Turkey; Prof. Pavisc, Jugoslavia; Prof. François, Belgium, secretary-general.

The European Society will hold a congress every four years, at two-year intervals with the International Congresses. The first congress will be held in Athens in April, 1960. The official report will be on "The pathogenesis and treatment of secondary glaucoma." The languages of all the member countries will be used.

Personals

Dr. Paul Boeder, former chief of the Bureau of Visual Science of the American Optical Company, has been appointed associate professor of physiologic optics in the Department of Ophthalmology of the State University of Iowa, Iowa City.

Dr. Arthur J. Bedell, Albany, New York, returned recently from Australia, Tasmania, and New Zealand. In Sydney and Hobart he gave a series of lectures and in Melbourne he spoke before the Australian Ophthalmological Society. During the sessions of the New Zealand Ophthalmological Society at Hamilton, he gave illustrated lectures on ophthalmoscopic subjects. Both the Australian and New Zealand societies elected Dr. Bedell an honorary member.



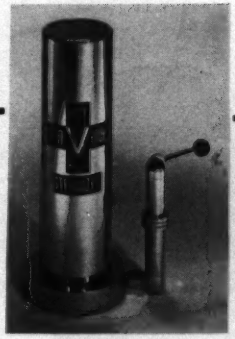
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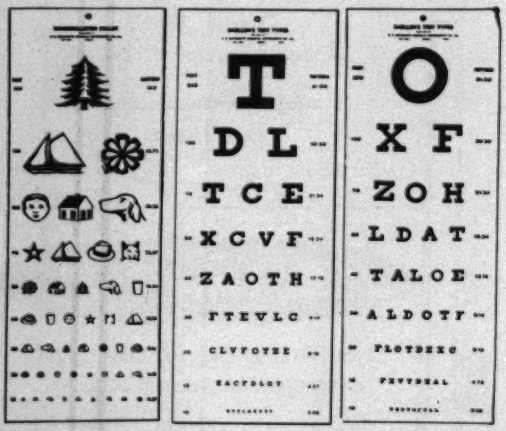
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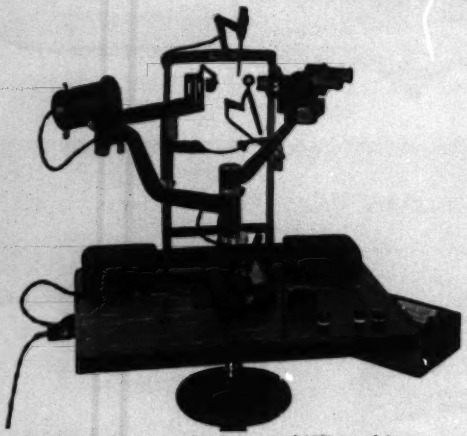
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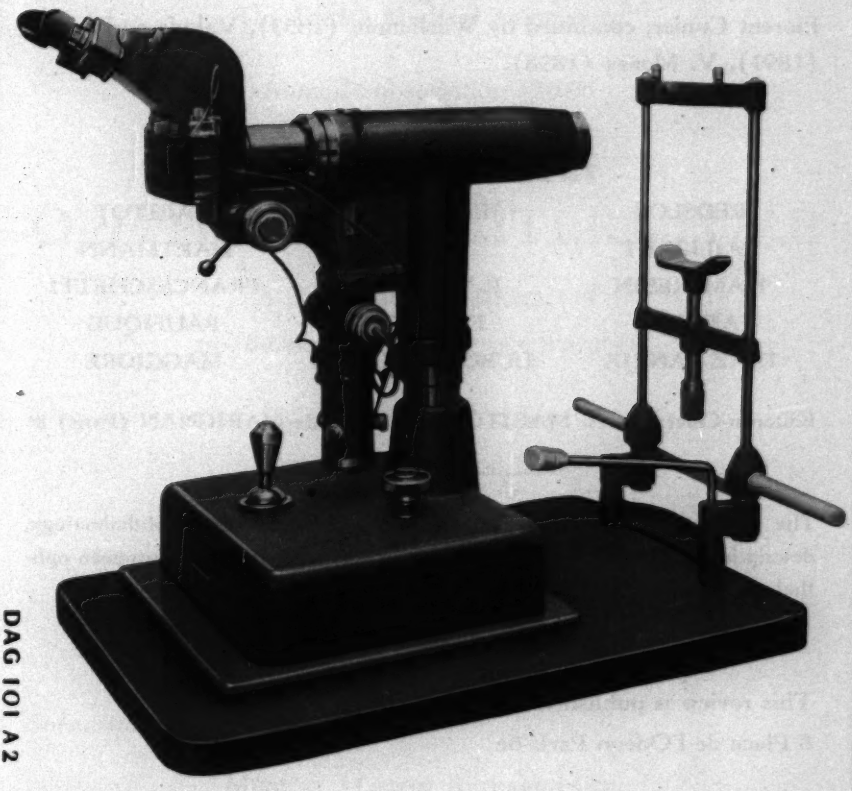
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